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ACUTE APPENDICITIS: ITS EARLY DIAGNOSIS.[1]

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IT may seem at first sight that the choice of such a subject as this for my paper tonight is not a happy one. It may be objected that acute appendicitis has been the best known of surgical diseases to medical men and to the laity for more than thirty years, and that everything that can be said about its diagnosis must have been said long ago.

If there are any of those present so complacent as to believe this, I would ask them to consider the implication of these following facts.

The Mortality of Acute Appendicitis.

I think it is universally agreed that if acute appendicitis is treated surgically in its earliest stages, the mortality is very low. In such cases, apart from the occasional tragedy of a pulmonary embolus or the rare occurrence of wound infections or chest complication, we can almost guarantee a speedy recovery to our patients. The necessity for early operation has been stressed by most of the surgical masters. "The first indication in appendicitis," said W. W. Keen, "is to call a surgeon." "The first stage of appendicitis," said Murphy, "is the accepted time for salvation, and likewise the time for removing the appendix." "Medical men," he said, "keep on treating appendicitis expectantly until the only thing left to expect is an autopsy; too often the surgeon receives the patient when he is a candidate for the efforts of the undertaker instead."

If the diagnosis of acute appendicitis can be accurately made in its earlier stages and surgical operation is expertly performed, we should expect by this time a very great reduction in the mortality rate of this disease.

[1] Read at a meeting of the Queensland Branch of the British Medical Association on July 3, 1931.

I think, in Australia at any rate, when a confident diagnosis of acute appendicitis is made, operation is rarely opposed by the laity. How far do statistics support these expectations?

In Table I is shown the mortality from appendicitis in Queensland. What can be wrong in Queensland? Do our great distances and sparse population militate against early operation and explain this increasing mortality and the still more ominous rising mortality rate? Perhaps English statistics will give a truer view of the position.

TABLE I.

Showing Mortality from Appendicitis in Queensland.

Year.	Males.	Females.	Total Number of Persons.	Rates per 100,000 of Population.
1910 ...	24	12	36	6
1919 ...	36	16	52	7
1921 ...	28	29	57	7
1922 ...	46	23	69	9
1923 ...	36	24	60	7
1924 ...	45	24	69	8
1925 ...	45	23	68	8
1926 ...	56	28	84	10
1927 ...	43	28	71	8
1928 ...	51	20	71	8
1929 ...	53	20	73	8
1930 ...	43	29	72	8

Table II shows the mortality from acute appendicitis in England and Wales. American results[3] are the same (see Graph I and Graph II).

TABLE II.

Showing Mortality from Acute Appendicitis in England and Wales.

Year.	Number of Deaths.	Population.	Mortality Rate.
1915	2503	35,390,000	7·1
1916	2603		
1917	2443		
1918	2316		
1919	2429		
1920	2531		
1921	2711		
1922	2735		
1923	2826		
1924	2756		
1925	2862	38,900,000	7·4

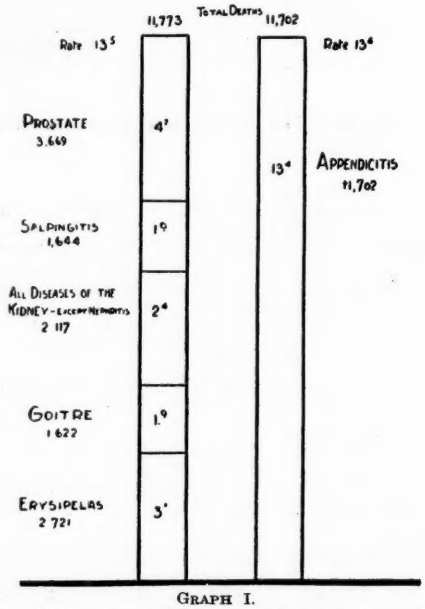
It is appalling to realize that the mortality rate of acute appendicitis is not falling; rather it is rising. The number of deaths annually from appenditicis in the United States of America equals all those from salpingitis, pelvic abscess, surgical diseases of the pancreas, spleen and thyreoid, gall stones and ectopic pregnancy combined. And yet early operation in appendicitis is now practised to a much greater extent than ever formerly.

TABLE III.

Showing Age Groups and Mortality in England and Wales.

Years.	Number of Deaths.
0-15	756
15-25	487
25-45	614
45-65	706
65-75	222
75 over	77

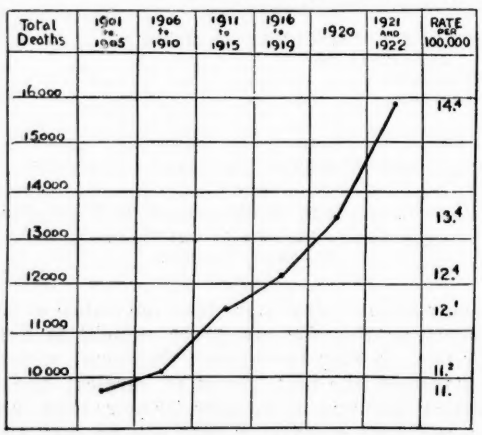
"The explanation of the mortality," says Wilkie, "is clear and permits of no doubt, namely, that the majority of the patients now operated on early are of the class which did not contribute to the mor-



GRAPH I.

(Reproduced from Willis's paper, *Surgery, Gynecology and Obstetrics.*)

tality, while in the fatal and dangerous type of case, operation occurs little, if at all, earlier than it occurred twenty years ago." This is a considerable blow to our complacency. Evidently our



GRAPH II.

(Reproduced from Willis's paper, *Surgery, Gynecology and Obstetrics.*)

knowledge of the diagnosis of appendicitis is not yet complete.

Age Groups and Mortality.

More even than the number of deaths, it is the age groups in which the deaths usually lie that constitute the tragedy of appendicitis.

TABLE IV.

Showing Mortality by Decades.

Age by Decades.	Cases.	Deaths.	Percentage.
Under 5	31	5	16·1
To 10	220	23	10·5
Second	954	51	5·3
Third	665	48	7·3
Fourth	276	44	15·9
Fifth	121	21	17·3
Sixth	67	22	32·8
Seventh	34	16	47·0
Over 70	4	4	100·0
Not stated ...	34	5	14·7

In Miller's series (Table IV) only 4·4% of the cases occurred in persons over fifty years of age, though 22·2% of the deaths were in these patients.

Although the total death rate from cancer is six times that from appendicitis, 80% of the deaths from appendicitis occur before the fiftieth year, while only one-fifth of the deaths from cancer occur before that age. Thus in the important working years of life appendicitis is almost as great a menace as cancer. Now, think of the vast organization and stupendous effort expended on the problem of cancer, the old man's disease, and then consider if we should not say as Murphy did twenty years ago, that "instead of it being time to stop talking about appendicitis, it is time to begin talking about it, and talking most emphatically and seriously about it."

So far I have endeavoured to show that the persistent high death rate in appendicitis is in part due to the fact that early treatment is missed because the practitioner has no fixed conviction about the early diagnosis of the dangerous types of appendicitis.

There is still another side of the question which is not so important from the point of view of mortality statistics, but which, from the standpoint of the relations between the medical profession and the public, is of vital importance. This lies in the frequent diagnosis and attempted treatment of acute appendicitis when the disease is not present. Every surgeon on the staff of a public hospital is aware that a large number of patients suffering from a great variety of pathological conditions, all having right iliac fossa pain or tenderness, are sent to the hospital with an accompanying letter stating that they are suffering from acute appendicitis. Thus Frazer, writing from the Edinburgh Royal Infirmary, stated that of fifty patients admitted with the provisional diagnosis of acute appendicitis, only twenty-six were actually suffering from this disease.

No one infers that this large percentage of error is due to wilful misdiagnosis; it is certainly not so. But the translation of such an error into private practice, with the unnecessary or misapplied operations resulting, is a grave matter.

Many writers have commented on a mistrust of the medical profession by the general public that has been growing in recent years, and it has been stated that nothing has done more to foster this mistrust than indiscriminate operating following the diagnosis of appendicitis. The general public cannot know that most of this misplaced surgery is due to an honest error in diagnosis; and it behoves us to see if these errors in diagnosis cannot be lessened before more serious harm is done. Though I have insisted that these wrongful diagnoses are not made with any dishonest motive, it is doubtful if the public can long be persuaded to think so.

It would seem, then, that the diagnosis of acute appendicitis in its earlier stages is not yet universally accurate; but rather that the more dangerous forms of appendicitis are still being recognized only when a dangerous stage is reached, while, on the contrary, a large number of miscellaneous conditions are being misdiagnosed as appendicitis.

I should like to sum up this part of my paper by the following extract from Dr. Jeff Miller's recent admirable article on appendicitis in *The Journal of the College of Surgeons of Australasia*:

> This is . . . a disease which is perhaps the best known of all diseases, certainly of all surgical diseases. This is a disease which in its typical form is so clear cut in its syndrome that even a lay person can diagnose it. This is a disease which is admittedly surgical and which is so simple in its earliest stages that the veriest tiro can handle it. This is a disease which would not carry a mortality of 1% if it were diagnosed promptly and operated upon with equal promptness. But the other side of the picture is not so bright. This is a disease which in a very large proportion of cases begins with symptoms which do not suggest the popular conception of it, and because of this fact it is treated by the laity, and too often by the profession, with ill-advised and dangerous remedies. This is a disease which is temporized and played with and bandied between physicians and surgeons, because they are medical cowards in face of a diagnosis which may be vague or difficult. Finally, this is a disease which, when it has passed beyond its first stages, offers problems of judgement and technique which tax to the utmost the resources of expert surgeons, and yet which, because appendicitis is supposed to be a simple disease and appendicectomy is supposed to be a simple operation, the merest neophyte in surgery feels himself competent to handle.

TABLE V.

Showing the Position of Five Thousand Appendices.

(Wakeley and Gladstone.)

Position.	Number of Cases.	Percentage.
Anterior or pre-ileal	47	0·94
Splenic or post-ileal	25	0·50
Pelvic, on psoas muscle near or hanging over brim of pelvis	1606	32·11
Subcaecal, beneath caput caeci	101	2·02
Post-caecal, or retrocolic	3219	64·38
Ectopic	2	0·04

The Mechanism of Abdominal Symptoms.

When we commence the study of abdominal disease, a problem in diagnosis has to be faced that does not concern us in most other parts of the body.

On the external surface of the body the application of a stimulus will result in the appropriate receptor receiving and transferring the stimulus to the brain, where it rises to consciousness. Not only will the character of the stimulus, but also its location, be accurately registered. In the gastro-intestinal tract we are faced with entirely different circumstances. Observations on exposed gut have

taught us that the common stimuli of touch, heat and pain are here not capable of eliciting any response from the organism. Evidently receptors for these stimuli are completely lacking in the intestinal tract. What stimuli, then, are capable of eliciting a response from the gut?

As far as is known certainly at present, the only stimulus of which the intestinal tract takes cognizance, is change in tension in the muscle fibres of its walls. Thus excessive distension of a gut lumen is capable of producing symptoms, as are excessive contractions in its walls, such as over-forcible peristaltic contractions or muscle spasms.

This lack of receptors for common stimuli is a very real bar to easy diagnosis. Take the process of inflammation for example. The phenomena of redness, heat, swelling and pain, which are all appreciated on the surface of the body and on which the diagnosis is based, are none of them capable of evoking local signs in the gut wall. Only by generalized symptoms, such as fever and malaise, and perhaps by disorder of local function, such as nausea or diarrhœa, can we suspect an inflammation confined to the intestinal wall. Only secondary effects, such as intestinal distension or spread of the inflammation to the parietal peritoneum, can help us to localize the site of such an infection.

This is the first important point to remember, that inflammation of the gut wall does not of itself evoke any direct local signs by which it may be recognized.

Tension stimuli in the intestinal tract, if of sufficient intensity, give rise to pain, either continuous or intermittent, according to the character of the stimulus. The next difficulty is that this intrinsic tension pain is not localized to the site of the stimulation, but is felt diffusely, and perhaps in quite a different locality from that occupied by the affected gut.

To understand why this is so, it is necessary to recall briefly the way by which the gut has reached its present position in the abdominal cavity. In early embryonic life the gut consists of a straight median tube, slung from the posterior abdominal wall by the dorsal mesentery. As growth proceeds, three distinct divisions can be recognized: the fore-gut, later to become the stomach, the hindgut, and, between them, the mid-gut. The mid-gut ultimately becomes the intestinal tract from the duodenum to the middle of the transverse colon. It is this part which concerns us. Owing to its rapid increase in size and because of the relatively enormous space occupied in the abdominal cavity by the liver in these early embryonic days, the mid-gut becomes partially herniated into the umbilical cord. From the post-arterial segment of the mid-gut the future caecum and appendix are growing out as a diverticulum. During this stage of rapid growth the two extremities of the mid-gut loop are becoming relatively closer to each other and the mesenteric attachment of the mid-gut loop comes to occupy quite a small space in the middle line corresponding to the area existing in the adult between the duodenum and transverse colon.

The mid-gut grows to such an extent in the umbilical cord that return of the physiological hernia *en masse* becomes impossible, and the actual return has to occur in sequence of gut, usually the upper coils returning first and the region of the caecal diverticulum last. Because of this return in sequence and because of the continued increase in length, the mid-gut loop becomes rotated and its coils gradually take up positions in all parts of the abdominal cavity. Later still these in part become glued by secondary adhesions to the positions that they have taken up.

But these fixations are merely secondary factors, and it must be emphasized that the mid-gut loop is primarily a median structure, and that its true mesenteric attachment is to the narrow space between the duodenum and the transverse colon. And thus you will see why the primary intrinsic pain produced by distension anywhere in the mid-gut loop is not felt at the site occupied by the stimulated loop, but it is felt over the site of the real attachment of the gut, that is, in the middle line, in the epigastrium above and around the umbilicus. And this is the second point to be noted: the intrinsic pain from tension stimuli in the mid-gut loop (and this includes the appendix) is a diffuse mid-line epigastric pain. This is intrinsic pain.

A sufficient stimulus can also produce what are called referred symptoms. It is well recognized that when one division of a nerve such as the trigeminal is stimulated with sufficient intensity, the response (in this case pain), which at first was strictly localized, gradually becomes diffused, so that ultimately the stimulus seems to be producing a response over a wide distribution of the nerve. Thus an aching tooth may end in an aching face and eye.

In a somewhat similar way sensory stimuli entering any segment of the central nervous system, if they are severe enough, may overflow into other nerves entering that same segment and sensitize these nerves. These sensitized nerves exaggerate the responses passing along them, so that touch becomes a painful hyperæsthesia, pressure becomes deep tenderness, and ordinary non-painful stimuli are registered as pain.

All these exaggerated responses are interpreted as coming from the peripheral distribution of the nerve concerned.

The sympathetic nervous system, the fibres of which carry the sensory stimuli from the intestines, sends connector fibres to the spinal cord between the sixth thoracic and the second lumbar segments; and sufficiently intense stimuli entering these segments by these connector fibres can overflow into the ordinary spinal nerves entering that particular segment and sensitize them.

In the appendix and caecum the segments commonly affected are the eleventh thoracic and occasionally the segment on either side of it. The referred symptoms so produced do not at present directly affect us in the diagnosis of appendicitis,

but they will be referred to more fully in considering the differential diagnosis.

Still a third mechanism must be studied before we are ready to consider rationally the diagnosis of appendicitis. I refer to what Alvarez has styled "the reverse peristalsis syndrome."

One knows that the passage onwards of intestinal contents is effected by what are known as peristaltic waves, which consist in a wave of relaxation followed by a wave of contraction, and that the normal effect of these waves is that the contents traverse the gut in a cephalic to caudal direction. Alvarez has propounded the question: why do the waves normally travel in the direction they do? And he has answered in this threefold way: (i) Peristaltic waves travel from a more irritable to a less irritable portion of the bowel, (ii) the degree of irritability of the bowel varies directly with the blood supply, (iii) normally the blood supply of the bowel diminishes steadily from the stomach to the colon. Since the activity and the irritability of the bowel increases gradually from the upper end of the bowel to the lower, Alvarez says that the food follows a gradient of muscular force similar to the gradient in the case of gravity which keeps water running down hill in a pipe line or stream. The gradient of forces in the bowel can be flattened or reversed in two ways: (i) by irritating the lower end of the bowel till it becomes as active as the upper end, (ii) by increasing the blood supply of any local area of gut till it becomes as irritable as the upper segment. Tension stimuli sufficient to elicit intrinsic pain are sufficiently intense to produce this irritation and thus produce reversed peristalsis, that is, travel of intestinal contents in a cephalic direction. Local inflammations produce the same result in a less intense manner. An early symptom produced by flattening or reversal of the gradient is nausea, while more marked flattening induces vomiting. A resulting effect is constipation.

So far we have dealt with mechanisms in the gut itself. Neither the visceral peritoneum nor probably the parietal peritoneum reacts to the ordinary stimuli; but outside the parietal peritoneum lies the layer of subperitoneal fascia which is as sensitive to stimuli as is the skin itself. A spread of inflammation from a viscus to this tissue produces an inflammatory condition which acts as an external inflammation does, that is, it produces pain and tenderness. The superjacent muscles over such an inflamed area are held tightly contracted, that is, rigid, to guard this area from external trauma.

Such parietal pain can be accurately localized, and when it becomes intense it seems to blot out the appreciation of coexisting intrinsic visceral pain.

Summarizing, then, this discussion of the mechanisms involved in appendicitis, we have explained the existence of: (i) intrinsic visceral pain, only produced as a reaction to tension stimuli and felt in the case of the mid-gut loop diffusely in the middle line above and around the umbilicus; (ii) referred symptoms, noted as hyperalgesia and hyperæsthesia over the peripheral distribution of certain intercostal nerves; (iii) parietal pain, produced by extension of visceral inflammation to the parietal subperitoneal tissues, and (iv) the reverse peristalsis syndrome, produced by irritation or inflammation in the lower portions of the gut and manifested as nausea or vomiting and constipation.

Varieties of Appendicitis.

From the standpoint of the diagnosis of appendicitis we must consider the appendix in its following aspects: (i) As a mucous membrane-lined diverticulum from the caecum; (ii) as an organ with an abundant store of lymphoid tissue in its walls; (iii) as a muscular tube which can normally empty itself of contents in the same way as the rest of the intestinal canal; (iv) as an organ in close contact with the parietal peritoneum. How close this contact is is not always recognized, but the summary by Wakeley and Gladstone of the position of five thousand appendices (Table V) will make this relationship clearer.[5]

Probably 96% of these appendices are in intimate contact with the posterior parietal peritoneum.

The continuity of the appendiceal mucosa with that of the caecum renders this organ equally liable to any inflammation that may affect these neighbouring portions of gut. The appendix wall is probably frequently inflamed under conditions that produce such functional disorders as diarrhœa, but the condition will usually be quite undiagnosable because, as I have shown, the intestine has no way of registering local signs of inflammation other than by disorders of function; also, if the inflammation is more intense, a flattening of the intestinal gradient may be produced, causing some nausea or a little vomiting. This is a true medical appendicitis and is no more the province of the emergency surgeon than are the associated enteritis or colitis, or such localized inflammations as produce an acute gastric or duodenal ulcer. Occasionally, as will be described later, a true surgical appendicitis may result, and this variety following on a colitis is sometimes described as a residual appendicitis.

The lymphoid tissue in the appendiceal wall is equally subject to recurrent inflammatory states with that in the rest of the body, and such conditions probably frequently occur. Young adolescents with chronically infected tonsils often get vague upsets in which a mild fever with nausea or a little vomiting is present, sometimes accompanied by tenderness on the right side in the lower part of the abdomen, indicating a slight spread to the sensitive parietal subperitoneal tissues. Such an attack is another variety of medical appendicitis. Even should a large area of tenderness or muscular rigidity be present, suggesting a larger degree of extra-appendiceal spread, this does not of itself point to the necessity for emergency surgical treatment. Such a condition will settle down, perhaps with the development of some peri-appendicular adhesions, and it is exactly analogous to the condition present during the exacerbations of a chronic duodenal or gastric ulcer.

In the same way, blood-borne infections from distant foci may settle in the appendiceal wall, but there again, while the condition remains a simple inflammation, it is quite unrecognizable as an appendicitis, and, further than that, such a condition can no more cause death than can any other uncomplicated inflammation in the gastro-intestinal tract. We see, then, that simple appendiceal wall inflammations are purely medical diseases and not surgical "emergencies."

What, then, is surgical appendicitis? What complication becomes added to an appendix wall inflammation that confers the special danger to life so that appendicitis is universally regarded as an urgent surgical condition? The danger is the danger of perforation with widespread sowing of infection and the development of general peritonitis, and it lies in the production of appendicular obstruction. Damage to the appendiceal wall is not sufficient; rise of tension within the lumen must be added to produce the danger.

Surgical appendicitis, then, is acute appendicular obstruction, and the diagnosis of surgical appendicitis is the diagnosis of acute appendicular obstruction.

Appendicular obstruction may be a direct or an indirect result of appendiceal wall inflammation. Directly it may occur from the swelling incident to the acute inflammatory reaction. When the inflammation has spread to the appendix from the caecum, the base of the appendix will naturally be most affected, and a blockage of the lumen will occur here more readily than at other parts. Thus a state of closed loop obstruction results with all its potentialities for danger.

Indirectly appendiceal wall inflammation acts as a predisposing cause to luminal obstruction in three ways: (i) Each recurrent inflammation leaves a certain amount of scar tissue in its train, and the result of this scar tissue must be a narrowing of lumen, so that in future attacks this portion is likely to become occluded with inflammatory swelling earlier than others. (ii) By peri-appendiceal spread an appendix wall inflammation may produce adhesions which in turn produce kinking of the appendix. (iii) By the widespread production of scar tissue in the appendiceal wall, the property of emptying which, in the character of a muscular tube, it normally possesses, is interfered with, and stagnation of contents with consequent absorption of moisture and the production of fæcoliths result. The presence of a fæcolith in turn predisposes the patient to local inflammation with rapid resulting blocking.

As the result of one of these factors or a combination of such factors, acute appendicular obstruction frequently supervenes upon simple appendiceal wall inflammation or occurs almost without inflammation as a result of fæcolith block. What are the dangers of such a blockage? The dangers are identical with those of any other closed loop obstruction of the intestinal tract.

A rapid filling of the closed loop with secreted mucus occurs, with progressive distension of the walls. Interference with the blood supply of the appendiceal wall is early in evidence, the pressure necrosis so produced being most marked where the internal pressure is greatest, as for example, over the impacting fæcolith. After a variable period of time, sometimes no longer than six hours, a gangrenous patch is present in the appendix wall and conditions are ripe for a burst through of the contents, with widespread soiling of the peritoneal cavity. Such has been a stage in the illness of nearly all patients who have ever died from appendicitis. The resulting peritonitis needs no description.

Very occasionally an appendix becomes distended with practically sterile mucus. In these circumstances, and providing the appendix wall still possesses expansile properties, an enormous degree of distension may result. I have once operated on a patient in whom the appendix resembled a distended intestinal loop, with walls so thin as to be transparent.

We are now brought to the stage where we recognize that the danger to life of appendicitis is solely the danger incident to acute appendicular obstruction. How, then, can appendicular obstruction be recognized and the patients with surgical appendicitis be discovered?

Our study of the abdominal mechanisms provides the answer. The body has provided receptors for the very stimulus, and for this only, that here exposes it to danger. The appendiceal distension is reacted to by the production of pain, felt where all intrinsic mid-gut pain is felt, in the epigastrium, near the middle line, above and around the umbilicus. Depending on the condition of the muscular wall of the appendix, that is, whether peristalsis is still capable of being elicited, we may in the earlier stages have either intermittent colicky pain or continuous distension pain. With cessation of the distension, as may occur with relief of the obstruction or bursting through the lumen, the pain, of course, ceases. The distension also elicits in greater or less degree the reverse peristalsis syndrome. Nausea and usually a mild degree of vomiting (once or twice) is produced, and constipation is the rule. In the extreme distension of which I have spoken previously, vomiting is often incessant, and in fact this condition exactly mimics acute intestinal obstruction. Summarizing now this portion of our paper, we see that appendicitis may consist of: (i) pure appendiceal wall inflammation not diagnosable as an entity, but the cause of some obscure fevers, the only local signs elicited being a mild degree of the reverse peristalsis syndrome—probably showing itself as nausea; or (ii) appendiceal wall inflammation *plus* peri-appendiceal spread, showing itself, if it causes sensitivity of the parietal subperitoneal tissue, by localized tenderness and soreness. These conditions are both pure medical entities and are to be treated as

other similar localized intestinal wall infections are treated.

The indication for emergency surgical treatment in appendicitis is the presence of acute appendiceal obstruction, either primary or secondary to appendiceal wall inflammation; and safety in appendicitis lies in operation before perforation has occurred.

Appendiceal obstruction is clinically recognized by the onset of intermittent or continuous diffuse mid-line epigastric pain, and perforation by its cessation with the subsequent development of general peritonitis and spreading parietal sensitivity.

The symptoms of surgical appendicitis are therefore as follows: With or without the prior occurrence of nausea, the patient is seized with persistent diffuse epigastric mid-line pain. He vomits once or twice soon after the onset. This pain either persists or, after a variable time, merges into or is replaced by a completely different localized pain in the right lower part of the abdomen. Tenderness is present over the area of the localized pain and muscular guarding (rigidity) may be present. As the appendix wall becomes more and more damaged by the infection, the intrinsic epigastric pain diminishes, and rupture of the appendix is marked by its abrupt cessation.

I have stated that an emergency operation need not be performed in appendicitis unless a clear and definite history of epigastric or umbilical pain in the early stages of the attack can be obtained. Does clinical experience support this view? I say emphatically that it does.

I have gone through the records of more than three hundred cases of appendicitis in which I have operated. In this series there were fourteen deaths. Every one of these fourteen patients had a history of diffuse epigastric or umbilical pain in the earlier stages except two, in whom the pain was felt diffusely across the lower part of the abdomen. These were both examples of gangrenous perforated pelvic appendices, and it is probable that the severity of their later parietal pain, felt, of course, lower down, tended to make them place lower the position of their primary pain.

Conversely, I am not aware that any patient to whom, because of the absence of appendiceal obstruction symptoms, I have refused operation, has ever died.

I have records of ten patients on whom, for various reasons, I have operated as an emergency measure, although no history of epigastric pain could be obtained.

None of these had an open perforation. In two cases, one a free pre-ileal appendix, the other a bound-down retrocaecal one, there is a special mention that no obstruction of the lumen seemed to be present, though the walls were acutely inflamed. In six cases the attacks were obviously recurrent, and firm adhesions bound the appendix into close contact with either the peritoneum of the lateral abdominal wall or the posterior peritoneum over the sacrum.

I think that in these cases, as in the rare retroperitoneal ones, the parietal infection pain is so early and so severe that it blots out the more diffuse and probably less acute generalized pain, just as it seems to do in the later stages of the ordinary attack.

In one case I operated on the eighth day for an appendiceal abscess, in which lay a gangrenous perforated appendix, in an intelligent man who stated that he had suffered no pain whatever, although for the early days of his sickness there had been an unpleasant feeling in the epigastrium. He insisted that this was not a pain, however. Finally, in only one instance have I found a free distended appendix when cross-questioning could not elicit any history of epigastric pain.

So I can say quite definitely that my experience supports the view I am enunciating, namely, that no patient is being exposed to danger of his life if emergency operation for acute appendicitis be refused, unless a clear history of diffuse epigastric or umbilical pain in the early stages of the attack is obtained. The emphasis in this statement is on the word emergency. After mature consideration many patients in this group will ultimately have their appendices removed.

What importance are we to attach to the other symptoms of appendicitis?

Temperature.—A knowledge of the patient's temperature is of no importance, except that fever above 38·9° C. (102° F.) is very exceptional. When we remember that the important pathological change is a mechanical one, an obstructive rather than an infective one, we should cease to look for an elevation of temperature as an aid to diagnosis. "Make the diagnosis first and then take the temperature" is still a sound dictum.

Pulse Rate.—The pulse rate also is of no value in the early diagnosis. The rising pulse rate of which the text books speak, indicates that the patient is rapidly becoming a candidate for the undertaker rather than the surgeon.

Leucocytosis.—Remembering again that we are dealing with a mechanical rather than an inflammatory condition, we should not be surprised to learn that blood studies are not reliable as an index to pathological change.

Albuminuria or Pyuria.—Never be misled by the presence of pyuria into missing the diagnosis of appendicitis. If the characteristic pain distribution is present, we are dealing with some type of mid-gut loop obstruction, though the urine be solid with albumin. Quite apart from the possible prior existence of a renal condition, appendicitis can cause urinary symptoms in these ways: (i) By referred symptoms along the lowest intercostal nerves, (ii) a pelvic appendicitis may irritate the bladder wall, (iii) a retrocolic appendicitis may irritate the ureteral wall. In this last condition a very marked degree of pyuria is frequently present.

Anomalous positions of the parietal pain and tenderness need cause no difficulty if the primary

importance of epigastric pain be realized. This never changes.

One other factor in the history is important when we are faced with a recurring attack of abdominal pain, and that is, the number and frequency of the attacks. It is rare for an appendicitis to recur at intervals of less than from two to three months, and it is extremely rare to obtain a history of more than six previous attacks of pain. Each attack leaves its residue of damage, and as a general rule the severity of the attacks gradually increases, so that by the fifth or sixth attack surgical intervention becomes imperative.

Differential Diagnosis.

The foregoing discussion of the varieties of appendicitis and the mechanism of the production of symptoms will have made it clear that there is only one major symptom that marks the presence of surgical appendicitis, and on this symptom the differential diagnosis must be based.

Epigastric pain is an early and a constant symptom. All others are inconstant and late. Any attempt to base a diagnosis on such late, indirectly produced signs as right iliac fossa pain and tenderness, as is done by almost every text book, can only involve us in a maze of difficulties. The region of McBurney's point can be likened to a cross-roads where the urinary, pelvic and gastro-intestinal systems converge, as well as being the site of referred symptoms from numerous distant foci, and a differential diagnosis based on tenderness in this region sets one the hardest of tasks.

With epigastric pain of the type described we are on very much easier ground. Such pain can only be due to tension stimuli somewhere in the mid-gut loop, and, practically speaking, the differential diagnosis becomes that of mid-gut obstruction or irritation, with the chance always greatly in favour of the appendix as the offending organ.

Strangulated Herniæ.—Strangulated herniæ must first be excluded. Remembering their possible occurrence is usually all that is necessary, although in fat people the decision as to whether an obstructed femoral hernia is present may be one of real difficulty.

Volvulus of the Small Gut.—I do not think that volvulus of the small gut is usually diagnosable from appendicitis, and in four cases I made the most likely diagnosis, namely, that of appendicitis. It is a closed loop obstruction just as appendicitis is a closed loop obstruction. In theory more distension might occur with the former and more local tenderness with the latter, but in practice the difficulty in differentiation is almost insuperable. The sound diagnostician will continue to diagnose the common as against the rare condition. The amount of vomiting is not a reliable sign; I have seen a gangrenous volvulus around a Meckel's diverticulum with almost no sickness, while the tensely distended appendix may cause incessant vomiting.

Meckel's Diverticulitis.—Meckel's diverticulitis is not likely to be distinguished from appendicitis, although the situation of the parietal tenderness might theoretically assist in the diagnosis.

Acute Intussusception.—Acute intussusception should usually be diagnosable. The age incidence, type of onset, occasional blood-stained mucous discharge, and the presence of the tumour make up a syndrome which will not be often missed.

Acute Enteritis.—Acute enteritis is only occasionally a difficulty. Remember that in enteritis the diarrhœa will follow closely on the pain and vomiting and will probably relieve them. An ætiological explanation of an enteritis may obtrude itself, and especially in food poisoning cases the existence of other sufferers will help in the diagnosis. A practitioner is not on sound grounds in urging immediate appendicectomy on a number of patients in the same house at the same time, though I have heard of this being done. Do not forget that as an enteritis settles, a residual appendicitis may remain and appendicular obstruction may supervene at any time.

If in the presence of abdominal pain and diarrhœa, serious difficulty is encountered in excluding appendicitis, I would advise that the patient be put to bed in a sitting position, starvation insisted on, and an adequate dose of morphine given. If the pain reappears after the effect of the morphine has worn off, regard the condition as surgical and treat it accordingly.

When faced with persistent epigastric pain it must next be considered whether this is of local parietal origin due to underlying inflammation. Pancreatic lesions, localized duodenal perforations and sometimes acutely inflamed gall-bladders provide such examples. But a careful history will almost always reveal essential differences, remembering that parietal inflammations are relatively late manifestations of intraabdominal disease, while the associated tenderness of a parietal inflammation is not present in the epigastrium in appendicitis.

Provided you base your differential diagnosis on the symptom of epigastric pain, you will be hard put to it to find any other difficulties in the diagnosis of appendicitis, except possibly one. I refer to the deliberate placing of the pain higher than it is felt by the sophisticated subjects of acute salpingitis, with the idea apparently of raising the status of their disease. I have met with one such case recently; and it is a very real difficulty, because, however certainly the physical examination reveals acutely inflamed tubes, yet the apparent presence of epigastric pain must lead us to diagnose a secondary appendicitis and act accordingly.

Although I think I have now considered most conditions which can really be mistaken for appendicitis, I am quite aware that I have scarcely touched on the diseases that in practice are so mistakenly considered. These, which include all those conditions with which right lower abdominal pain and tenderness are associated, and not necessarily in order of frequency, are as follows:

By far the most common are the recurrent attacks of pain which cannot be described more shortly

than the right lower abdominal crises of right-sided visceroptosis. Once understood, such a condition is unmistakable. One obtains a history somewhat as follows:

The patient is usually a girl in her late adolescence, perhaps twenty years of age. She is well built, does not look very ill, however long her attack may have been progressing, and usually shows none of the external stigmata associated with mid-line ptosis. This is not her first attack of pain, but, on the contrary, during perhaps two years she has had many such attacks, perhaps during one or two days every three weeks. Although she has had so many attacks, she has probably never consulted a doctor, and with the possible exception of the present attack, there has been no increase in their severity. One finds that all the attacks, like the present one, have consisted of exclusively right lower abdominal pain, usually felt along a strip just above the inguinal ligament in front of the anterior superior spine. Vomiting has probably occurred and a temperature of 37·8° C. (100° F.) may be present. The most striking point on physical examination is the extraordinary tenderness in the right iliac fossa, apparently so acute that the patient scarcely tolerates the most gentle palpation. There is an associated tenseness of the anterior abdominal muscles, rather easily distinguished from the rigidity of parietal inflammation. Even though the pain has been present several days, no mass can be palpated in the abdomen.

If the abdomen of such a patient is adequately explored, the same condition will invariably be found, namely, a normal appendix attached to a tense, large, thin caecum with a complete absence of the normal lateral fixation of the ascending colon and caecum. The proximal large gut in these cases can be taken and lifted often six inches or more out of the abdominal incision.

Such pain and hyperæsthesia as I have described are due to a referred intercostal neuralgia of the eleventh and twelfth intercostal nerves, and I believe the actual mechanism of the production of symptoms is as follows: In the first place the lack of adequate lateral fixation of the ascending colon slows down the passage of intestinal contents in two ways: (i) by mechanically reducing the efficiency of the peristaltic contractions, and (ii) by the drag of the free caecum and colon on the weak mesentery setting up sympathetic nervous irritation with resulting bowel spasms which further delay the intestinal passage. The net result of these two factors is that the patient suffers from constipation and caecal stagnation, with recurring attacks of caecal distension. In the patient who gets these pains, a further condition is present. This is an abnormally easy transference of impulses which in themselves are not sufficient to give rise to intrinsic pain to the parietal intercostal nerves. In other words, a facilitation of the transference of these stimuli takes place. The result is that such a person, when she becomes subject to constipation and caecal distension, gets these seemingly severe attacks of intercostal neuralgia referred by the nerves concerned to their peripheral distribution.

Such patients are not subjects for operation. The pain usually recurs within two months of appendicectomy, while as far as my follow-up reports of a few cases go, colopexy does not give satisfaction in this type of visceroptosis, though in certain other syndromes of right-sided ptosis it is a miracle-working operation. Treatment lies in keeping the caecum light, and in preventing this too easy transference of stimuli by such agents as bromides, and in giving the patient confidence, because it is very noticeable that after appendicectomy the patients do not worry about their attacks as they previously did. The knowledge that the attacks cannot now be the dangerous appendicitis they previously feared seems sufficient to make them quite bearable.

Especially in younger patients, a localized soreness attributed to appendicitis is often due to inflamed ileo-caecal glands. These patients are usually of the scrofulous type; there is a probable history of bowel upsets, the pain has persisted for a number of days without the development of a mass, there is no rigidity, and the condition generally is obviously a mild one. Such patients should not be subjected to operation. Abdominal filariasis, one example of which I have explored on the mistaken diagnosis of an appendiceal abscess, is a subgroup of the above.

Urinary conditions, and especially ureteral calculi, have often been confused with appendicitis. There is really very little resemblance. Except that they have the common meeting ground of right lower abdominal tenderness, their pain distribution and histories are quite dissimilar. It is much more important not to make the opposite error, and because of pyuria to miss the diagnosis of a retrocolic or pelvic appendicitis.

Pelvic adnexal conditions are very rarely, in my experience, a serious difficulty in the differential diagnosis. The physical signs are here so definite and the history so typical that a mistake is difficult to make. A pelvic appendix, however, is frequently inflamed by direct spread from such a pelvic peritonitis, and the occurrence in such a condition of epigastric pain is an urgent indication for operative interference.

Ovarian endometrial cysts sometimes offer difficulty, and their adhesion-forming properties may involve a pelvic appendix. The minor catastrophe of their rupture, with a consequent spread of parietal pain perhaps as high as the epigastrium, may be difficult to distinguish from a hyperacute onset of appendiceal obstruction; but the history of the increasing menstrual discomfort and the discovery of a pelvic mass are all distinguishing features.

Especially in past years, many cases of perforated duodenal ulcer were operated on under the mistaken diagnosis of appendicitis with generalized peritonitis. In such a case it is not the presence of board-like rigidity that is the distinguishing mark of a perforated ulcer, but rather the history of an

overwhelmingly sudden intraabdominal catastrophe, with the acute onset of pain so severe that the patient possibly fainted with the intensity of the shock. The rigidity supervenes almost immediately and is quite unlike the deliberate spread of a peritonitis in the later stages of an appendicular obstruction.

Such a condition as a perforated duodenal ulcer serves admirably to illustrate a thesis that has formed the basis of my paper, namely, that in all intraabdominal diagnosis it is the history that matters rather than the physical signs.

Compared to the knowledge of the position and type of pain and the sequence of events, any information that can be obtained by physical examination is of relatively little value.

It is not because a patient demonstrates the physical sign of widespread rigidity of his abdominal musculature that we diagnose a perforated ulcer—such a sign may be seen in many states, varying from visceroptosis to tetanus—but because we obtain a history of a sudden abdominal catastrophe, followed by a widespread occurrence of parietal pain.

Likewise, in appendicitis it is not the physical sign of right lower abdominal tenderness to which we should pay heed. Such also occurs in a multitude of conditions. But if a patient gives a history of diffuse epigastric pain which has persisted for several hours, we can say with absolute certainty that he has an obstructive or irritative condition in the mid-gut loop and that the chances are ninety-nine to one in favour of obstructive appendicitis, which demands an urgent abdominal operation.

Conversely, in the absence of the history of such pain, whatever the physical signs, it may be said that a condition requiring emergency appendicectomy is one of the last conditions that should be thought of.

References.

(1) Communication from Registrar-General's Office, Queensland, April, 1931.
(2) J. B. Hunter: "The Mortality of Acute Appendicitis," *The British Medical Journal*, March 19, 1927, page 508.
(3) A. M. Willis: "The Mortality in Important Surgical Diseases, Especially Appendicitis," *Surgery, Gynecology and Obstetrics*, Volume XLII, March, 1926, page 318.
(4) C. J. Miller: "A Consideration of the Mortality of Acute Appendicitis, with Special Reference to 239 Fatalities," *The Journal of the College of Surgeons of Australasia*, Volume III, July, 1930, page 40.
(5) R. J. Gladstone and C. P. G. Wakeley: "The Relative Frequency of the Various Positions of the Vermiform Appendix, as Ascertained by an Analysis of 3,000 Cases," *The British Journal of Surgery*, Volume XI, January, 1924, page 503.

ACHLORHYDRIC DYSPEPSIA.[1]

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IN the consideration of dyspepsia as a symptom complained of by the patient, it is necessary that the physician obtain a clear idea in his own mind of the patient's complaint when he labels it "indigestion," and, secondly, that he should have some clearly defined idea as to the various ætiological factors which may cause these symptoms, in order that some conclusion which will be of benefit to the patient in his treatment, may be arrived at.

There are few symptoms that require more of what may be termed the eliminative process of reasoning for their elucidation than does dyspepsia.

The symptoms referred by the patient to what he terms his stomach resolve themselves from the purely physiological standpoint into three main groups: (i) Secretory, those caused by excessive or diminished gastric secretion; (ii) sensory, due to stimulation of the sensory nerve supply of the stomach, and varying from slight degrees of discomfort to severe pain; (iii) motor, hypotonia, resulting in dilatation and dropping and excessive muscular activity, resulting in spasm, with or without vomiting, sensation of fullness *et cetera*.

It is not any one symptom, but the combination of symptoms complained of, that is of value as a guide to the cause of the dyspepsia. Pain in the region of the stomach may not be due to a gastric lesion at all; for example, the gastric crises of *tabes dorsalis* and vomiting may be due to a pregnancy or be of cerebral origin, as in *tumour cerebri*.

When it is determined that the symptoms complained of by the patient are caused by some upset of gastric function as outlined above, the first consideration is to exclude organic disease of that viscus, particularly ulcer of the stomach or duodenum, malignant disease, and pyloric obstruction from whatever cause, and, secondly, to exclude associated abdominal disease, chiefly chronic inflammation of the appendix and gall-bladder (with or without calculus), which may cause "reflex" dyspepsia. When these organic abdominal lesions have been excluded by a careful history and examination of the patient, combined, if necessary, with laboratory tests and radiological investigation, there still remains a large group of cases in which digestive upsets occur.

It is the weeding and sorting of this large residual group that presents great difficulty. In this group the operative causes are legion, and, excluding acute gastritis, which can scarcely be classed as a dyspepsia, the group constitutes the bulk of the dyspepsias which one is called upon to treat in practice. For the purpose of this paper chronic gastritis is included for consideration in this group, as it is impossble in a good many instances to decide on symptoms alone just where the so-called functional dyspepsias end and chronic gastritis begins, although the latter shows definite organic changes in the gastric mucosa and is associated with excessive production of mucus. From an ætiological viewpoint the difficulty is even greater.

A large proportion of these cases owe their origin to general disease outside the stomach, the dyspepsia being a secondary symptom, caused either by a chronic gastritis following the primary disease or by functional upset of the stomach activity. Such are the dyspepsias associated with pulmonary tuberculosis, genito-urinary disease, hyperthyreoidism,

[1] Read at a meeting of the Victorian Branch of the British Medical Association on July 18, 1931.

cirrhosis of the liver or myocarditis with cardiac failure.

When such general diseases as have just been enumerated have been in their turn excluded, there yet remains a balance of cases, manifesting digestive discomfort of varying degree, requiring treatment—the simple or functional dyspepsias. Problems of diet, rate of eating, teeth, focal sepsis, anxiety, overwork and worry are chief among the numerous causative factors, and it must ever be remembered that a dyspepsia that is at first labelled as simple, may in time become one which falls into one of the previous categories. A hyperacidity, if uncontrolled, may give rise to gastric or duodenal ulceration, especially in the presence of overlooked focal infection, whilst an achlorhydria may be a potential case of pernicious anæmia, and carcinoma may supervene upon any form of chronic dyspepsia; in fact, according to Hurst,[1] it never arises otherwise.

And further, outside disease which by reflex disturbance may be the cause, may at first be overlooked, later symptoms proving the presence of appendiceal or gall-bladder disease. Similarly, it is not infrequent for the earlier symptoms of pulmonary tuberculosis to be almost entirely referred to the impaired digestion, and only later, when the apical signs in the lung become more pronounced, is the true nature of the dyspepsia revealed.

The real test of correct diagnosis lies in the patient's maintained improvement under correct treatment.

Clinical Records.

During the past two and a half years, since the opening of a hospital at Yallourn, with laboratory and X ray facilities, we have obtained records of ninety-three patients with dyspepsia who received treatment, and some of these were further examined by the fractional test meal, by barium meal and by X rays. An analysis of these ninety-three cases along the lines suggested in the introductory remarks to this paper discloses the following distribution:

A. Gastric carcinoma (proven cases)	1	(inoperable)
B. Gastric and duodenal ulceration—		
(*a*) Perforation	1	(duodenal)
(*b*) Severe hæmatemesis	3	(duodenal 1, gastric 2)
(*c*) No complications	2	(duodenal—one had had previous gastrostomy)
C. Gastric and duodenal ulceration—suspected and treated as such (not proven)	11	
D. Chronic appendicitis—dyspepsia cured by appendicectomy	9	
E. Gall-bladder disease—		
(*a*) Operative cases	3	
(*b*) Medical cases	14	
F. Dyspepsia due to general causes, including chronic gastritis and the functional dyspepsias—		
(*a*) War neuroses, with gas	4	
(*b*) Abdominal migraine	2	
(*c*) Alcoholic gastritis	1	
(*d*) Patients with variable degrees of discomfort to pain, flatulence *et cetera*, relieved by alkali, and not included in any previous group	26	
(*f*) Hypochlorhydria	4	
(*g*) Transient achlorhydria	2	
(*h*) Achlorhydria	10	

In all, twenty-two patients were examined by the fractional test meal, and of these ten showed complete absence of free hydrochloric acid. One of these was a patient with pernicious anæmia and was shown this afternoon as a clinical case. Three are being treated as being suspected of early pernicious anæmia. One is a potential sufferer from pernicious anæmia and the remaining five show no evidence of the disease. Of the remainder, two manifested complete absence of acid on one occasion and lowered acidity on another; four had hypoacidity; three had normal free acid and three hyperacidity (a rise above 60 maximum free acid being present).

From this analysis it will be noted that sixteen of the twenty-two patients examined by the fractional test meal showed diminution or absence of acid. This in no way implies that this percentage, 17%, of the ninety-three patients alone suffered from such absence of free acid. It is more probable that many of the patients presenting the gall-bladder type of dyspepsia would have shown similar results. Hutchison[2] points out that in one series of a considerable number of patients with gall-bladder disease who were investigated by the test meal, hypoacidity of the gastric contents occurred in 77%, and the investigators concluded also that the hypoacidity was not the result of the gall-bladder disease, but a primary condition favouring infection of the bile passages.

The test meals were not carried out in any spirit of research, but simply because the patients concerned were not improving under simple treatment, consisting of regulation of diet, habits *et cetera* and removal of septic foci, and usually combined with drug treatment, such as compound bismuth powder or some similar alkaline preparation, and it was considered advisable to find, if possible, the cause for the lack of improvement.

Typical Case Histories.

The four case histories which follow are illustrative.

CASE I.—H.H., a male, aged forty-two years, was first seen on April 10, 1928, complaining of an irritable rash which he had had for two weeks. Examination disclosed a generalized urticarial rash, chiefly confined to the chest, abdomen and thighs. There was also constipation. *Lotio calaminæ* relieved the rash, and the constipation was treated by general dietetic measures and a mixture containing sulphate of magnesia (acid).

By April 17, 1928, the rash had quite gone, but the constipation was still troublesome. Three teeth which had been filled, were suspected of sepsis; the suspicion was confirmed by X ray examination and the teeth were extracted.

On August 25, 1929, he was seen again at the onset of an illness resembling influenza. His temperature was 38° C. (100·4° F.), his tongue was very coated. The heart, lungs and abdomen showed nothing abnormal on examination. Next day the temperature was still raised and his body was covered by a vesicular urticarial rash. He had had some looseness of the bowels with abdominal colic. Inquiry elicited the fact that on several occasions prior to April 10 he had had similar rashes.

This illness lasted ten days, the highest temperature reached being 39·2° C. (102·6° F.). There were several attacks of diarrhœa and a good deal of anorexia. The tongue was heavily coated for the whole period. General examination revealed nothing abnormal outside the gastro-intestinal tract throughout the illness, and no cause was discovered.

Dr. Ivan Maxwell saw him later and performed certain intradermic tests, a slight sensitivity to house dust and linseed being discovered. However, a test meal revealed complete absence of free acid in the gastric juice, pepsin was normal and there was no mucus. On his return this result was confirmed. An examination of the central nervous system revealed nothing abnormal. Blood examination revealed 4,000,000 red cells per cubic millimetre and a normal film.

Since then, October 21, 1929, his treatment has consisted of four mils (one fluid drachm) of dilute hydrochloric acid with each meal and 15 grammes (half an ounce) of "Heparidine" daily.

On September 8, 1930, the test meal still revealed achlorhydria, and the blood film was normal. On May 11, 1931, these findings were confirmed. The red cells numbered 5,000,000 per cubic millimetre. He has had no urticaria since the attack on August 25, 1929, and feels well and improved in general health.

CASE II.—Mrs. L.B., aged thirty-seven years, was first seen on March 22, 1929, when she complained of epigastric pain with nausea, retching and occasional vomiting. She had had a similar attack four years before and a second attack one year previously. She had been married six years and had no children. There was no loss of weight. The pain commenced immediately after food was taken, lasted some hours and often recurred at about 11 p.m. On other occasions there were days of nausea, but no pain.

Examination showed fullness and a moving mass in the right iliac fossa, the mass later proving to be the caecum distended with fæces. There was definite tenderness in the upper part of the abdomen on the right side. Pelvic examination revealed a normal uterus and adnexæ. Chronic cholecystitis was suspected and she was dieted accordingly and given alkali after food. For a time she improved.

On January 23, 1930, she was again seen. The dyspepsia was almost as troublesome, though she was gaining weight. The same abdominal fullness was present, and she complained of an umbilical discharge. The cause proved to be purely local. Treatment by diet and alkali was continued.

On June 27, 1930, she was seen at the onset of an attack of acute epigastric pain. She stated she had been getting worse for some time, vomited nearly every second day and had great discomfort and feeling of distension if she took a meal of any unusual size. This closely resembled her original attacks nearly six years before. She also had headaches and a feeling of blocking in the ears. The pain on this occasion was more severe, began immediately after meals and often lasted well into the night. There was no pain between the scapulæ or over the right shoulder. Examination disclosed definite tenderness to pressure over the upper part of the abdomen, in the centre, and also below the ninth right costal cartilage on deep pressure and respiration. Her temperature and pulse rate were normal, the tongue coated, but the breath clean. On her admission to hospital for investigation alkali was used to relieve her distress without effect. The test meal revealed complete achlorhydria with a trace of mucus. X ray examination after a barium meal revealed no ulceration of stomach or duodenum, the stomach being elongated and having fallen below the iliac crest in the upright posture. Treatment was as follows. She was sent home to rest, small, easily digestible meals were ordered to be taken every four hours, followed by two hours' rest on the right side to facilitate gastric emptying. Dilute hydrochloric acid in doses of 1·2 mils (twenty minims) was given with each meal, and bromides to insure mental rest. When she got up after two weeks' rest, she was ordered an abdominal support and abdominal exercises. In two weeks she was able to go away for a holiday. At the end of three months she was so well that she discontinued all acid with her meals and has had no symptoms of her trouble since then. A test meal done on July 10, 1931, revealed a normal gastric acidity with a maximum of forty units one and a half hours after the meal.

CASE III.—R.D., a male, aged fifty-six years, was first seen on November 6, 1928, complaining of pain in the central epigastrium and precordial area over a period of ten years and latterly associated with palpitation. There was no general association of the discomfort with the taking of food, though it frequently followed meals. Constipation was a troublesome feature; he was often depressed and his feeling of well-being was gone, and he slept badly. His position was one of many responsibilities, entailing much worry each day, which he could not shake off in the evening.

His appendix was removed in 1907. A test meal done ten years ago revealed hyperacidity and, until recently, alkali had relieved his symptoms. Occasional perineal pain for fifteen years was another feature which seemed to be due to the constipation and associated with hæmorrhoids. He had no urinary symptoms such as might be referred to an enlarged prostate.

Examination revealed a pulse rate of 80 and a normal response to exercise. His systolic blood pressure was 172 and his diastolic pressure 102 millimetres of mercury. The cardiac apex beat was situated 11·25 centimetres (four and a half inches) from the mid-line in the fifth interspace. The cardiac dulness was normal. The specific gravity of the urine was 1·025. No albumin or sugar was present. The lungs and abdomen were clear. On rectal examination no prostatic enlargement was noticeable, but hæmorrhoids were present. The teeth were sound.

Worry and work, with constipation, were looked upon as the chief causative factors in this case, and the patient was treated accordingly.

On November 4, 1930, he was seen again, suffering from pain in the right shoulder and right side of the chest, with peculiar paræsthesia, which later developed into a typical *herpes zoster* of the fourth, fifth and sixth thoracic segments. At this time he had definite dyspnœa on exertion, there was increased indigestion after food, fullness, but no pain or vomiting. He had lost some weight. Alkali did not relieve him. His condition was investigated at hospital with the following results.

On November 20, 1930, a test meal revealed a complete achlorhydria, no mucus being present. Blood urea was estimated as 45 milligrammes per 100 cubic centimetres of blood. A barium meal revealed nothing abnormal with the stomach. The hæmorrhoids were treated by the injection method and he was given two mils (half a fluid drachm) of acid with all meals three times a day.

A second test meal, given on December 4, 1930, still revealed achlorhydria, and this was confirmed on July 9, 1931. This patient did not always obtain relief by acid and there were occasions when alkali afforded him more benefit.

It is interesting to note that whereas ten years ago he had a definite hyperacidity, he now has an achlorhydria. He has slowly improved in general health in the past twelve months and has lost his depression. It is felt that his dyspepsia is a functional one, due to his work, and in the absence of mucus in all specimens tested, not due to a chronic gastritis, as was thought originally.

CASE IV.—Mrs. S., aged forty-five years, had had twelve children. The youngest child was born two and a half years ago, when it was found she had gross pyorrhœa and she soon afterwards had all her teeth removed. For years she had had a sallow anæmic appearance.

For the past six months, as soon as she took food she experienced a general discomfort in the left epigastrium and xiphoid angle. The condition became more painful half an hour after the meal and persisted for a considerable time. Lately the pain kept her awake at night and was more severe than in the day time. Meat, cabbage or potato made it worse, and at first it was relieved by alkali, but not latterly. There was no vomiting at any time. Constipation was becoming worse, but the motions were normal in appearance. Micturition was normal. Menopausal symptoms were present.

In the past three months there was considerable loss of weight, from 69·3 kilograms (eleven stone) to 56·7 kilograms (nine stone). She had enjoyed perfect health until she experienced a severe influenzal attack shortly after the birth of her last child.

Six months ago she had a severe attack of acute infective arthritis, being twice in hospital. Erythematous nodules were present on all extremities and on the abdominal wall. Since then she has been quite free from all joint pains, but her dyspepsia dates definitely from that attack and has been progressively worse since then. She was not seen again till two weeks ago, July 1, 1931, when her cachectic appearance, coupled with her loss of weight and gastric symptoms strongly suggested the possibility of a gastric carcinoma. She was admitted to hospital.

Examination revealed no tumour in the epigastrium. The liver was enlarged downwards two finger-breadths and its edge was smooth. The spleen was not palpable. Heart, lungs and central nervous system appeared normal. A test meal proved the presence of a complete achlorhydria, no mucus or bile being present. The blood film was normal in appearance; the hæmoglobin value was 75% and there were 3,900,000 red cells per cubic millimetre, giving a colour index of 0·9. Screening with X rays after a barium meal revealed a hypertonic stomach, fixed high up. Peristalsis was normal, but there was marked pyloric spasm, no meal leaving the stomach after the lapse of half an hour. It was thought there was a definite filling defect in the pyloric antrum, and a film was taken.

At exploratory laparotomy performed on July 14, 1931, a normal stomach was seen; it was explored by a pyloric gastrotomy through the pyloric ring. The liver was normal, the gall-bladder was normal and free from calculus.

It is remarkable that such evidence of apparent organic disease of the stomach could be produced by an achlorhydric dyspepsia, and it is proposed to treat this patient with acid and liver extract, as though she were suffering from a definite pernicious anæmia.

In the following general discussion will be presented some of the features of these sixteen cases, with conclusions therefrom.

Ætiology.

From the ætiological point of view practically all causes of dyspepsia may result in an achlorhydria, but some of these have a special tendency so to do. These causes are: (*a*) general diseases which tend to produce a chronic gastritis; (*b*) all general diseases of the asthenic type, associated with loss of weight, anæmia or cachexia, producing diminution of all gastric function; (*c*) chronic gastritis itself, which, though in its earlier stages may be associated with hyperacidity, tends sooner or later to give rise to excessive mucus in the stomach, with consequent diminution of the free gastric acid; (*d*) general and focal sepsis; (*e*) functional dyspepsias, caused by faulty habits of eating, diet *et cetera*, or lack of proper teeth; (*f*) worry, overwork, anxiety and the neuroses.

Why is it that in some subjects these causes produce a lowering of the gastric secretion, while in others there is an increased acidity? Hurst[3] explains this peculiarity of response as a difference of diathesis. He says that approximately 20% of all people have a tendency to digestive upset, 10% having the misfortune to be born with what he calls the "hypersthenic" gastric diathesis, and 10%, equally unfortunate, inherit what he terms the "hyposthenic" diathesis. Both diatheses are compatible with perfect health, but neglect of the means of avoiding indigestion is likely to cause it in those predisposed. If hyperchlorhydria develop, gastric or duodenal ulceration is likely to result. If, however, the hyposthenic type develop indigestion, hypochlorhydria will result, and, later, an achlorhydria. This type never suffers from ulcer formation, as for this the presence of free acid is essential.

Achlorhydria must be distinguished from achylia, which is the absence of all gastric secretion. It probably occurs only as a congenital condition (according to Hurst in 3% to 4% of all people), as the last stage of a chronic gastritis (atrophic gastritis) or in the latest stages of a carcinoma of the stomach. It may be symptomless, but, in the presence of any of the causes of indigestion, symptoms similar to achlorhydric gastritis may develop. The absence of pepsin as well as of all acid is the chief distinguishing feature. "Achlorhydria and *achylia gastrica* together constitute the one essential predisposing factor in the causation of Addisonian anæmia and subacute combined degeneration of the spinal cord" (Hurst).[4]

Symptoms.

The outstanding feature of the gastric symptoms in these cases is their variability and indefiniteness. Often there are no gastric symptoms at all, but a general feeling of lack of well-being, general unfitness, lassitude and depression, with a strong tendency to the development of neuroses, especially in females.

Pain is usually absent, discomfort and fullness being more prominent.

Flatulence is a common symptom, and often is aggravated by the use of alkalis, whilst air-swallowing may become a habit. Vomiting is not common, though nausea may be intense, both being aggravated by the excess of mucus which is present in the more marked cases of chronic gastritis.

The tongue is often coated. Diarrhœa may be present, especially in true achylia, when undigested muscle fibres may be found in the stool, but constipation is the rule. Anæmia of varying degree will often be found, and there is frequently loss of weight.

Pathology.

It is proposed to discuss only the physiological pathology of the upset of secretory and motor functions of the stomach and to leave untouched that of the numerous causative or associated conditions.

Secretory Changes.

Secretory changes are those demonstrated by the test meal. Complete absence of free gastric acid is

found. The total acid content was not determined in any tests done. Pepsin is also absent when *achylia gastrica* is the lesion or in the latest stages of carcinoma and atrophic gastritis. Mucus of varying amount may be present and is indicative of chronic gastritis.

Motor Changes.

Alteration in motor function is best demonstrated by means of the barium meal and X ray screen. If there be loss of muscle tonus, the stomach is frequently fallen in position, elongated and often dilated, and does not contract completely around its contents, the meal dropping from the œsophageal orifice to the bottom of the stomach with a splash effect. In the upright posture the lower margin of the stomach may be below the pelvic brim, resulting in delayed emptying.

In other cases there is not loss of tone, and even the reverse, a spasm of the stomach musculature. This was seen to a remarkable degree in two of the cases of achlorhydria. One is accustomed to associate such spasm with excess of free acid rather than with the reverse. In both cases the cachectic appearance of the patients and loss of weight suggested carcinoma. One patient whose case history has been given, was subjected to laparotomy, but no growth was found. In the other case the spasm was relieved by hypodermic injection of 1·3 milligrammes (one-fiftieth of a grain) of atropine. The blood films of both patients are normal in their appearance, though both patients have a definite secondary anæmia, proved by red cell count and hæmoglobin estimation, and are receiving treatment with liver as well as acid.

Diagnosis.

As has been stated in the introductory paragraph of this paper, the diagnosis is entirely eliminative. In practice, however, every patient with dyspepsia presenting for treatment cannot be given the consideration necessary to eliminate the host of general diseases with which dyspepsia may be associated. The exact nature of the individual symptoms which the patient collectively labels "indigestion," and their time relation to the taking of food, together with the appearance of the patient as regards colour, loss of weight *et cetera*, are the facts of prime importance.

Assuming that the major organic lesions of the stomach and duodenum, gall-bladder and appendix are excluded as far as is possible by the history and examination of the patient, the next problem is to discover, if possible, the cause or causes of the dyspepsia. It is necessary to inquire into possible errors in the diet, habits, work, worries and mental outlook of the patient, and to examine thoroughly the teeth, tonsils and other possible foci of infection. Frequently no satisfactory cause is discovered.

In the absence of X ray and test meal facilities, the diagnosis can be furthered by the therapeutic test. There is a general tendency to treat all dyspeptic patients by the administration of alkali, usually through the medium of compound powder of bismuth or some modification of it, and in most cases the expectancy is justified by its results, but it must be recognized that in a number of cases this will fail, and obviously when the gastric acidity is below normal or absent. If no response to alkali treatment be obtained after a fair trial, suspicion that this latter state is present should be aroused.

There seems no reason why the test meal should not be used more than it is by general practitioners, at least as a qualitative test, the addition of one or two drops of Topher's reagent in 5% solution to each specimen collected by the Rehfuss tube giving a bright red coloration if any free acid be present. If greater accuracy be desired when the test shows the presence of free acid, then titration may be done for a quantitative estimation.

Hutchison,[5] quoting Dr. Ryle, states:

> Dr. Ryle found the test meal of no value in the investigation of dyspepsias other than those in which there was gross organic disease of the stomach. In functional disorders, whether primary or reflex, the results were so varied that he believed the method to be really not worth trying.

While this may be quite true from the point of view of diagnosis of the cause of the dyspepsia, it is not true as regards the question of treatment, where the knowledge of the absence of free acid is of definite value in the drug treatment of the disease, as well as being a warning of the possibility of the preanæmic state.

Assuming that the therapeutic test of the failure of alkali to relieve symptoms, combined with the result of the test meal, has proved an achlorhydria, especially when more than one test has been made to confirm the result, it is essential that due consideration be given to the possibility of two important pathological conditions—gastric carcinoma and pernicious anæmia, including the preanæmic state and subacute combined sclerosis of the spinal cord.

For the exclusion of carcinoma a barium meal may be of real value. (Reference has already been made to fallacies in interpretation caused by spasm of the musculature of the stomach.) But, when any doubt arises, an exploratory operation must be performed, especially when pain and loss of weight with cachexia are present.

With the exclusion of pernicious anæmia the problem is even more difficult. There is no laparotomy to give a definite decision, and it must be realized that there are many stages between simple achlorhydria, through all degrees of the preanæmic state, to the well developed clinical picture of a typical pernicious anæmia, and if Hurst's view of the ætiology of Addisonian anæmia be accepted, then all achlorhydrics are potential sufferers from pernicious anæmia.

Examination of the blood film and estimation of the percentage of hæmoglobin and the red cell count, giving the colour index of the blood, are of vital importance and, moreover, it is necessary to do repeated tests at regular intervals. The halometer

may then be of value. Use may also be made of the Van den Bergh test.

Of equal importance is examination of the central nervous system for early signs of subacute combined degeneration of the spinal cord, which again must be done at regularly repeated intervals. Spinal cord degeneration may precede the anæmia and be easily overlooked. Of similar significance are the changes in the mental attitude. Depression and loss of mental energy which occur not uncommonly in primary anæmia, were noted in many cases of this series.

Prognosis.

Most of these cases respond excellently to treatment by dilute hydrochloric acid, the most troublesome being those in which there is a well developed alteration in the mental state.

When there is suspicion, from the symptoms and appearance of the patient, combined with blood examination, that the preanæmic state exists, the prognosis must be more guarded and governed by the results of treatment controlled by periodical examination of the blood and central nervous system. Four cases in the series are included in this group.

Treatment.

Treatment, to be effective, must be directed first against the cause, where this is possible. It is necessary to insure that the teeth are in good order and sufficient in number, so that the food can be masticated properly. Septic teeth, tonsils and antra should be eradicated, if present. Worries, anxiety and overwork should be removed as far as this is possible, especially if nervous symptoms be present, and, if the blood pressure be low, rest should be enforced.

Diet.

There is no need for the restricted dietaries that are necessary in the treatment of ulcerative conditions of the stomach. Certain principles guide one in advising the patient as to diet. First, the food should be taken as dry as possible to minimize dilution and consequent diminished concentration of acid available for gastric digestion. Secondly, the taking of excessive protein is unwise, except in so far as its aroma stimulates appetite, for it needs more acid for the initial stages of its digestion than does carbohydrate. Thirdly, fatty foods and those cooked in fat should be avoided, as fat, by absorbing free acid, inhibits digestion.

As constipation is usually present, the diet should have a high fruit and vegetable fibre content, allowance being made for the exclusion of certain vegetables, cabbage for example, which the patient finds by experience tend to promote flatulence. Paraffin oil is a useful adjunct in the treatment of the constipation. The preparation of the food is important, as its palatability and appearance are helpful in stimulating gastric secretion. When the sensation of fullness or distension is marked, relief will be obtained by taking smaller, easily digestible meals at shorter intervals.

Drugs.

Hydrochloric acid is the specific drug in the treatment of these patients. For guidance in the administration of acid Beckman[6] recommends, among others, the following principles: (i) Give the acid in as large amounts as possible compatible with the tolerance of the patient, up to 10 mils (2·5 fluid drachms) per day. (ii) Use the acid in fractional doses, commencing during the meal and continuing through digestion for at least half an hour. (iii) A meal of carbohydrate only, given with the full dose of acid, is valuable in that it induces a condition of free gastric acidity.

Bromides combined with various glycerophosphate syrups have been used in those cases where the nervous symptoms loom large, while *liquor atropinæ sulphatis* is being given before food in the two cases showing marked pyloric spasm.

Where a definite anæmia exists, iron and arsenic may be added to the mixture containing acid, and liver, 180 grammes (six ounces) per day, or its equivalent in reliable brands of liver extract, is added to the dietary. "Heparidine" is one preparation which has been tried.

In functional cases and cases of mild gastritis, when the cause has been removed, there is often a return to normality of the gastric acidity, and all treatment may be discontinued, a cure having resulted. But, when the repetition of the test meal proves the continued absence of free acid, it is necessary to continue the administration of dilute hydrochloric acid, maybe for life, and in the preanæmic cases this applies to the liver dietary also.

When, associated with the general asthenic state, there is definite gastroptosis, confirmed as it may be by barium meal and X ray screening, the wearing of a properly fitting abdominal support is advisable and plays no small part in relieving digestive discomfort, as well as the constipation. To this end exercises for the abdominal muscles, as well as general exercise, walking, golf *et cetera*, will lead to improvement and help to improve the mental symptoms.

Summary.

1. There are many cases of dyspepsia, often overlooked, in which the test meal reveals diminution or absence of free hydrochloric acid in the gastric juice.

2. The therapeutic test of administration of acid is of value in those cases which do not give an early and maintained response to alkali treatment when organic and reflex causes for the dyspepsia have been excluded.

3. Confirmatory test meal, at least by the qualitative method, is useful as a guide to treatment.

4. It is important to recognize the relation of achlorhydria to the preanæmic state and the early spinal cord changes of Addisonian anæmia.

Acknowledgement.

In conclusion I wish to thank Dr. Francis, Resident Medical Officer at the Yallourn Hospital, for his invaluable assistance in collecting the material for this analysis and carrying out the necessary laboratory and radiological tests.

References.

(1) A. F. Hurst: "Chronic Indigestion," *The Practitioner*, January, 1931, page 84.
(2) R. Hutchison: "The Medical Annual," 1931, page 285.
(3) A. F. Hurst: *Loco citato.*
(4) A. F. Hurst: *Loco citato.*
(5) R. Hutchison: "The Medical Annual," 1931, page 208.
(6) H. Beckman: "Treatment in General Practice," 1930, page 423.

"PERNOCTON" IN OBSTETRICS.

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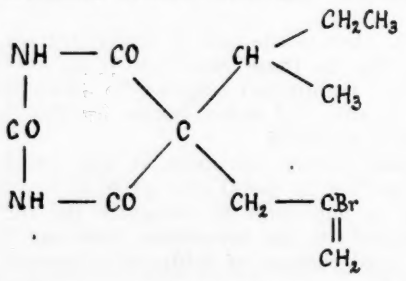
THE desiderata in obstetrical anæsthesia are those of general anæsthesia with important additions. These additions are: (*a*) The anæsthetic should not interfere with uterine contractions; (*b*) if possible, it should permit reflex abdominal muscle contractions; (*c*) it must not affect the fœtus. Until the advent of "Pernocton" it could not be said that such an anæsthetic, with the partial exception of nitrous oxide, existed.

The extent of anæsthesia is determined by the concentration of the anæsthetic in the blood and tissues, and the appropriate concentration is such that the cortical cells are depressed, but the medulla, with its respiratory and cardiac centres, is not so affected.

It is customary to produce anæsthesia by the use of volatile anæsthetics which reach the blood through the pulmonary alveoli. Blood concentration of the anæsthetic is readily controlled by regulating the concentration of the volatile anæsthetic in the inspired air, that is, by drop or other method. The ease with which a general anæsthetic, produced by the use of volatile anæsthetics, may be controlled is the most important advantage attaching to their use.

I am indebted to Professor C. Stanton Hicks, of this University, both for clinical information which he obtained in Europe last year, and for the actual samples of "Pernocton" which he brought from Germany for my use.

"Pernocton" is a 10% solution of the sodium salt of secondary butyl-β-bromallyl barbituric acid.



It is a white crystalline substance soluble with difficulty in water, but readily in alcohol.

Since it contains an asymmetric carbon atom, "Pernocton" is a racemic mixture of two optical isomers.

Animal experiment has shown that acetonyl barbituric acid is the fragment of the "Pernocton" molecule which is excreted in the urine, and this represents only one-fifth of the amount administered, that is, the remainder is destroyed in the body. Moreover, an animal can be fed with acetyl acetonyl barbituric acid with the result that only a small portion can be recovered from the urine, pointing to the fact that the molecule can be oxidized by the organism. "Pernocton" thus, by the introduction of the $-CH_2-CBr=CH_2$ group, acquires one of the most desirable attributes of a good hypnotic, the property of rapid decomposition or alteration to non-toxic substances within the tissues.

Another significant structural advantage is the presence of the secondary butyl radicle which by its asymmetric carbon atom simulates naturally occurring molecules. It is well known that the potent natural molecules, for example, adrenalin, hyoscyamin, have asymmetric molecules and that the lævorotatory form is twelve to eighteen times more active in the latter instance. Since the proteins of the cells also contain such asymmetric structures, it might be anticipated that racemic mixtures would show a greater specific affinity for the protoplasm of certain nerve cells.

The toxic margin with "Pernocton" is very high; intravenous administration to rabbits gives a ratio of 1:10 for average narcosis dose. Intravenous injection must be slow in order that (*a*) the concentration of the drug in the blood be not suddenly raised, and (*b*) the hypertonic "Pernocton" solution at a pH of 9·5 be thoroughly mixed with the blood.

Intravenous administration is desirable in that it admits of absolutely accurate dosage and rate of absorption by the nerve cells. Intramuscular or enteral administration is uncertain in this respect and, although necessary when more dangerous compounds are used, it is not important in "Pernocton" anæsthesia, except perhaps for additional amounts which are given intramuscularly mainly because of the slower absorption.

Its initial use was tried in asylum practice, and our first clinical knowledge of its value is due to Bumm,(1) head of the surgical clinic at the Charité, Berlin. Bumm's first review of one hundred cases begins the literature upon this subject. Since then (1927) until April 1930, 200,000 cases of clinical administration have been reported.

In the cases under consideration I have followed the Continental experience (*vide infra*), and two cases are given in order that the technique used might be known, and also to demonstrate the necessity for inducing by suggestion a satisfactory mental attitude in the patient. The dose of "Pernocton" is one cubic centimetre per 12 to 15 kilograms of body weight, the rate of injection being 0·75 cubic centimetre per minute.

CASE I.—Mrs. E.H., aged twenty-four years, a *primipara*, weighed 54 kilograms. On examination the cervix was dilated "five shillings" at 4.45 p.m. The pulse rate was 86. At 4.50, 3·3 cubic centimetres of "Pernocton" were slowly injected intravenously. The patient was asleep before

the injection was completely given, but, owing to a large number of onlookers who disturbed the psychic effect, appropriate suggestion was not obtained. At 5 p.m. two cubic centimetres of "Pernocton" were given intramuscularly. The pulse rate was still 86. The uterine contractions appeared to increase and at these times the patient became most restless. At 8 p.m. the patient was extremely restless, and 1·5 cubic centimetres of "Pernocton" were given intravenously and 1·5 cubic centimetres intramuscularly. At 10.15 p.m. I delivered the patient, using forceps, but owing to the restlessness a few drops of chloroform were inhaled through a mask.

Amnesia was complete from the first intravenous injection, and the patient stated on different occasions that she recalled nothing.

This case is mentioned as indicating the faulty technique, which did not observe the necessity for absolute quietness and adequate suggestion. I find that the best results occur when I am left alone with the patient.

Case II.—Mrs. H.L., aged thirty-two years, a *primipara*, weighed 66 kilograms. On examination the cervix was dilated "five shillings." The pulse rate was 82. The patient was kept very quiet and was told that she would soon lose the pains and fall asleep. There were no onlookers. "Pernocton," 3·8 cubic centimetres, was very slowly administered intravenously. After 2·5 cubic centimetres had been given, the patient was asleep. An intramuscular injection of 2·2 cubic centimetres was made ten minutes after the intravenous injection. The condition was excellent, there was absolute quietness between contractions, with some facial registration of pain during the contractions. Some manipulation was required later, and forceps were applied without any trouble at all. The birth of the infant was followed in twenty minutes by delivery of the placenta, the pulse rate then being 92. The patient subsequently remembered nothing and progressed most favourably. The patient herself, a *primipara* of thirty-two years, considered that she had a very good confinement.

The difference in these two cases, which are so alike in many of the features, is to be found in the technique of quietness and suggestion. The method of administration now adopted was taken from a paper by Brammer,[2] of the Universitäts Frauenklinik, Freiburg im Baden, whose fifteen hundred "Pernocton" deliveries have been completed, and of these he has especially analysed 225 cases to determine the best procedure. In the same paper is given a minute analysis of the results of "Pernocton" anæsthesia as used in other clinics, and he indicates especially the following:

Goldschmidt[3] states that 3·5 cubic centimetres is the lowest amount that suffices for a case.

Vogt[4] uses 8·8 cubic centimetres as an initial intravenous injection and after forty-five minutes gives four cubic centimetres intravenously in addition. He says there are no ill effects.

Mütz[5] gives 3·5 cubic centimetres intravenously and 1·5 cubic centimetres intramuscularly. He says that anæsthesia lasts for three hours.

Brammer concludes that two points must be strictly observed: first, the slow injection which obviates any vomiting, and, secondly, the correct method of suggestion.

From his series of cases Brammer has given the following as a satisfactory ratio between the initial dose and the succeeding dose:

58% were given 3·5 cubic centimetres intravenously and 1·5 cubic centimetres intramuscularly.
10% were given 3 cubic centimetres intravenously and 2 cubic centimetres intramuscularly.
8·6% were given 4 cubic centimetres intravenously and 1 cubic centimetre intramuscularly.
2% were given 4 cubic centimetres intravenously and 2 cubic centimetres intramuscularly.

He then states that, using five cubic centimetres of "Pernocton," it was found that the intravenous route was many times more efficacious than the intramuscular method; he considers that the latter has a variable rate of absorption. He further advises that in *primiparæ* the "Pernocton" should be given when the os is "five shillings" dilated; the cervix in *multiparæ* should be dilated only "three shillings."

The average duration of the "twilight" sleep for the single dose in 72 women was found to be 2·53 hours, and the average duration in 28% was 1·45 hours. In 80% of Brammer's cases the anæsthesia lasted for three hours and the amnesia was complete. One case was found to be quite refractory, but no "Pernocton" death was recorded. In eighteen infants there was some degree of respiratory distress, but this was regarded as being due to a coexisting maternal toxæmia.

For the purpose of giving a second injection where labour is at all prolonged, the relationship of the second to the first injection must receive careful consideration. Brammer recommends the following:

First Dose.		*Second Dose.*	
Intravenously.	Intramuscularly.	Intravenously.	Intramuscularly.
4.0 c.cm. ..	2·0 c.cm.	2·0 c.cm. ..	2·0 c.cm.
3·5 c.cm. ..	1·5 c.cm.	1·5 c.cm. ..	1·5 c.cm.
3·0 c.cm. ..	1·0 c.cm.	1·0 c.cm. ..	1·0 c.cm.

Conclusions.

1. "Pernocton" is a suitable obstetrical anæsthetic.
2. "Pernocton" has been abundantly tried and fulfils all the requirements of obstetrical anæsthesia.
3. The results which I have obtained correspond very closely with those published in Europe.
4. Quiet surroundings and careful suggestion are essential to the technique.
5. The intravenous route is the more exact method of injection, while the more slowly absorbed intramuscular injection is used for additional doses to maintain the concentration of the anæsthetic in the blood.

Acknowledgements.

I desire again to record my thanks to Professor Hicks; also to Dr. Brotchie, who helped with cases at the Queen's Home Maternity Hospital.

References.

(1) R. Bumm: *"Intravenose Narkosen mit Barbitursäurederivaten,"* *Klinische Wochenschrift*, Volume XVI, 1927, page 725.
(2) H. Brammer: *"Versuche mit einer besonderen Form des reinen Pernocton Dämmerschlafs in der Geburtshülfe,"* *Schmerz. Narkose, Anaesthesie*, Volume II, 1931, page 399.
(3) R. Goldschmidt: *"Der Einfluss des Pernocton auf den Geburtverlauf,"* *Zentralblatt für Gynäkologie*, Volume VI, 1930, page 344.
(4) E. Vogt: *"Erfahrungen mit Pernocton beim geburtshilflichen Dämmerschlaf,"* *Medizinische Klinik*, Volume I, 1928, page 24.
(5) H. H. Mütz: *"Pernocton-Dämmerschlaf in der Geburtshülfe,"* *Zentralblatt für Gynäkologie*, Volume XLIV, 1928, page 2819.

THE USE OF HEAVY METALS IN THE TREATMENT OF CANCER: A PROGRESS REPORT.

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In two previous communications[1][2] the author has given a *résumé* of the investigations he has undertaken in the administration of heavy metals in malignant conditions. The metals used were administered in the form described by the author,[3] in which the metal was combined with the envelope

of red corpuscles. This method gave a preparation which could be standardized and was stable. Some of the preparation, which had been kept in the ice chest for more than two years, still has the same microscopical appearance and physiological effects when injected into the laboratory animal as when first prepared. The metals used have been bismuth, lead, copper, uranium in various combinations. In the pursuit of this investigation the author has had to rely in the main on patients who come to him in his private practice and in a small gynæcological clinic at the Women's Hospital. A few patients are also from time to time allotted to him at the Austin Hospital, Melbourne (where patients are generally sent when the attending practitioner feels that their condition is incurable and hopeless), but not until recognized methods of surgery, X rays or radium therapy have been exhaustively tried. Most of the last class of patients are in the last stages, and as their expectation of life is rarely more than a few weeks, they constitute very poor material on which to make any deductions as regards the value of the method. In the present communication it is the author's desire to record the further history of three patients, two of whom were referred to in previous reports.

CASE I.—The patient, number 10 of both reports, was first seen on February 1, 1928. She was forty-six years of age and had had radium applied (apparently for inoperable cancer) in January, 1927. Pus and blood were coming from vagina and rectum. Some hæmaturia was present. Bismuth-lead injections were commenced on February 5, 1928. Her weight was 55·3 kilograms (eight stone eleven pounds). She was very ill. On December 25, 1928, her weight was 69 kilograms (ten stone thirteen and a half pounds). On June 2, 1931, her weight was 66·4 kilograms (ten stone seven and a half pounds). She looks and feels well. There is no discharge. Vaginal examination reveals an atrophic vault of the vagina.

CASE II.—The patient, number 37 of the second report,[2] suffering from epithelioma of the urethral orifice, was seen in July, 1929. She had had a urethral caruncle snipped off by a doctor previously. Recurrence took place. The caruncle was "nipped off" for biopsy. Epithelioma was reported. On July 3, 1929, injections were commenced. A copper-lead preparation was used. She felt ill for about two months afterwards, but is now very well. She was last seen on July 1, 1931.

CASE III.—The patient was seen on June 5, 1930. She had had a growth on the breast for nine or twelve months, with discharge and bleeding from the nipple. An ulcerating, fungating mass was adherent to the skin and chest wall. Large glands were present in the axilla; they greatly impaired the mobility of the arm. The patient had a cough. The first injection of bismuth-uranium-lead preparation was given on June 8, 1930. On March 21, 1931, she looked remarkably well. The breast was scarred and clean. The arm was mobile. The patient had had no other treatment except the injections referred to.

Although only three cases are here recorded, these results, from the nature of the cases, are sufficiently encouraging for the writer to hope that some surgeon, not satisfied with the results obtained by present methods, will be prepared to give metallic therapy a place in the treatment of early and operable cancer. The author has treated operable and borderline cancers with injections as well as by the recognized methods, but sufficient time has not yet elapsed for reports of these to be of any value.

Sampson Handley[4] found that when he combined radium therapy with operation in cancer of the breast, his "five years cures" jumped from 25% to 40%.

Suppose a series of, say, one hundred patients were given the metal therapy just before and after operation, with or without radium or X ray therapy, the patient would not be deprived of the chance that surgery and radiology give, and one would have the opportunity of determining whether these injections would be effective in limiting the development of metastases which already exist in unrecognizable form in many so-called "operable" cases.

To expect therapy to perform miracles when the patient is already exhausted by the general effects of the growth, sepsis, operation and ray therapy, is manifestly not giving the method a chance. Could not this investigation be carried out at one of our established clinics with the usual scientific controls?

References.

(1) J. Leon Jona: "The Treatment of Inoperable Cancer by the Injection of a Colloidal Preparation of Various Metals—Bismuth, Lead, Copper," THE MEDICAL JOURNAL OF AUSTRALIA, November 10, 1928, page 587.

(2) J. Leon Jona: "Corpuscular Medication, A New Method of Administering Heavy Metals and Other Elements: The Method Applied in the Treatment of Inoperable Cancer by the Administration of Lead, Copper and Bismuth: A Progress Report," *Transactions of the Australasian Medical Congress* (British Medical Association), Third Session, 1930, page 456.

(3) J. Leon Jona: "A New Method of Administering Heavy Metals, with Reference to the Treatment of Cancer," *The Lancet*, July 7, 1928, page 15.

(4) Sampson Handley: "Parasternal Invasion of Thorax in Breast Cancer and Its Suppression by Use of Radium Tubes as Operative Precaution," *Surgery, Gynecology and Obstetrics*, December, 1927, page 721.

AN OCCASIONAL CAUSE OF TRIGEMINAL NEURALGIA.

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ONE occasionally encounters cases of facial neuralgia in which the trigger point is in the mucous membrane overlying the alveolar margin of the mandible or maxilla, where a tooth has been previously extracted. This was suggested to me first by Sandes, of Sydney, some five or six years ago. Since then I have carefully observed these cases, and on going into the past history of many of the patients have elicited a story in some of difficult tooth extractions with, presumably, laceration of the gum.

In the resultant scar tissue nerve fibres may become entangled and we have a condition allied to "painful scar," but with this difference, that the scar is in a position where pressure is being frequently exerted upon it, particularly during the act of mastication. In my experience this is not the whole story. There is another superadded factor, namely, the presence of a thinned-out, almost knife-edge alveolar margin over which the mucous membrane is stretched tightly, making the condition comparable to "conical amputation stump."

Patients are generally able to locate the pain to a small area; from this it radiates to other branches of the fifth nerve. Pressure upon the spot is painful, so is mastication for the same reason. For the conical stump of an amputation one is usually able to give relief by removing some of the bone, supplementing that in many cases by removing the end of the nerve (on which there is usually a neuromatous enlargement) and turning it back upon itself.

In the class of case under consideration I excise an oblong-shaped portion of mucous membrane down to the bone; one generally extends the incision both anterior and posterior to the painful area so as to be sure. The bone being exposed and being generally knife-edged, as I have previously explained, it is nibbled with gouge forceps; this readily allows the edges of the wound to be approximated and sutured with fine chromicized gut.

A general anæsthetic is desirable, with the use of a sucker to take away the blood in the mouth. Of course, the operation could be done under narco-local anæsthesia.

Pathological examination of the removed tissue in some cases reveals evidence of neuroma, in others not; but it would need a more exhaustive examination than is generally undertaken before one could say definitely that neuromata were absent.

The result of this operation, whether I am right or wrong in the pathology of the disease, is extraordinarily good. In the five or six cases in which I have done it, there has been wonderful relief; temporary in one case in which I removed the soft tissues without touching the bone; more or less complete when both procedures have been carried out. One of the recent cases has been a disappointment; I had to go on to avulsion of the inferior dental nerve just above its entrance into the mandible; at present the patient is well and free of pain.

I have a vivid recollection of the first case in which I did it.

It was on a woman, and was very successful; I showed her at a clinical evening at the Sydney Hospital; the husband came along as an exhibit also; he said home had been a very miserable and unhappy place for all the family prior to the operation.

I have had occasion to see this patient recently; relief was complete for twelve months; since then she has had some pain occasionally.

Examination shows a spot very tender to pressure over the anterior portion of the edentulous mandible; it is uncertain whether this was included in the area which I excised. She is to come into hospital to have it removed. I feel confident this will complete the cure.

Reports of Cases.

PRIMARY EPITHELIOMA OF THE SCROTUM.

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I AM indebted to my former house surgeon, Dr. John Osborne, for the notes on the following four cases of primary epithelioma of the scrotum which occurred in my hospital practice in the last six years. Several factors are of interest. Three of the patients had been workers in coal or coke for periods from twelve to twenty years, two were stokers, while the third was a coke worker. The remaining patient had been a sheet metal worker in fine dust most of his life.

The second feature is that all patients have a commencing history of trauma superimposed upon the longer history of chronic irritation from fine particles; and a third is the presence of all four cases on the left side, while all four patients "dressed" on the right, that is the growth occurred in the moist angle between scrotum and medial surface of the thigh.

CASE I.—J.P., aged sixty, was for twenty years a fireman in a bottle works. He had a fungating growth the size of a hen's egg on the left side of the scrotum which arose from an abrasion received seven months before. The inguinal glands were enlarged. The growth, together with the glands, was excised and sent to Dr. Keith Inglis for examination, who reported squamous epithelioma without glandular involvement. There has been no recurrence four years later.

CASE II.—P.F., aged fifty-five, was a sheet metal worker in fine metal dust for many years. He had a red lump the size of a pea on the left side of the scrotum with surrounding area of hyperkeratosis of three months' duration following an abrasion from a kick. The inguinal glands were enlarged. The growth and glands on both sides were excised. They were examined by Dr. Keith Inglis who reported squamous epithelioma without glandular involvement. Four years later there was no recurrence.

CASE III.—J.B., aged forty-three, was a coke worker all his life. He had a small circular ulcer at the left lower extremity of the scrotum following trauma some months before. Glands were palpable on both sides. The growth, together with both inguinal glands, was excised. Dr. Inglis reported primary squamous epithelioma without glandular involvement. Six years later there was no recurrence.

CASE IV.—Wm. J., aged sixty-three, was for twenty years a stoker at sea. He had a circular raised growth of six months' duration the size of a shilling on the left side of the scrotum immediately opposite the perineal band of a hernia truss. Inguinal glands were not enlarged. The tumour was excised and later examined by Dr. Keith Inglis, who reported squamous epithelioma. The patient had an uneventful convalescence.

THE USE OF VACCINE IN PERTUSSIS.

By ELLEN M. KENT HUGHES, M.B., B.S. (Melbourne),
Armidale, New South Wales.

As the Commonwealth pertussis vaccine is perhaps still only on trial with many medical practitioners, I thought it would be of interest if I recorded my experiences with it. I found it to be of great benefit during an epidemic in 1925-1926 and again during an epidemic at the end of last year. I always use the simple pertussis vaccine and give one cubic centimetre of "B" strength (500 million organisms per cubic centimetre) as the initial dose, 0·6 or 0·8 cubic centimetre of "B" strength (5,000 million organisms per cubic centimetre) for the final dose. Three or four injections are given at intervals of five days and there have been no disagreeable sequelæ in any instance.

When the vaccine is used for the treatment of whooping cough, the spasmodic cough and vomiting generally lessen in severity immediately, even if the whoop is well established. If the child has the paroxysmal cough, but no whoop, the latter generally develops immediately after the injection of the vaccine; there is but little accompanying vomiting and the whoop becomes less after about one week and finally disappears within three weeks.

I have had satisfactory results when the vaccine treatment was applied prophylactically. Generally the school children in a family become affected first, and I then inject vaccine into the babies and toddlers. In a number of instances these contacts have escaped the disease altogether. The parents themselves are now recognizing the use of the vaccine, and during the last epidemic several children were brought to me for treatment to prevent whooping cough from developing. I have treated about eighty children and adults with pertussis vaccine; and although this is not a large number, it is sufficiently large to show that the vaccine is a valuable remedy for this distressing complaint. The only patients whom it failed to relieve were two infants aged one month and three months respectively, suffering from well established pneumonia. These children both died.

I shall quote a few instances of children treated with pertussis vaccine at the end of last year.

D.M., aged five years, had a whoop that had been present for fourteen days; she had vomited all her food for three days and coughed continually night and day. On examination the child was found to have an accompanying bronchitis. After the first injection of vaccine the vomiting stopped; after the third the cough ceased entirely at night. Her brother, aged two years, and sister, aged seven weeks, were given injections and neither developed the disease.

P.C., aged two years, had a whoop that had been present for one week, and vomited several times a day. The mother came to me on account of her three weeks' old baby to whom she wished protective injections to be given. The baby did not develop whooping cough and the little boy recovered rapidly.

S.A., aged four years, had been whooping for three days. After injection of vaccine his condition improved rapidly, and his brother, aged two years, who had also been given an injection, did not develop the disease.

J.O., aged seven years, B.O. and G.O., both aged six years, had all been whooping for one week. After administration of vaccine J.O. and G.O. recovered rapidly; but B.O., a very highly-strung child, vomited for ten days. A brother, aged two and a half years, developed a slight attack of whooping cough; the baby, aged one year, escaped.

J.L., aged thirteen months, had been whooping for two weeks, and had been very wheezy for the two previous days and exhausted after spasms of coughing. On examination scattered rhonchi and râles were heard over both lungs; the temperature was 37·58° C. (99·6° F.); the pulse rate was 110 and the respirations 36 in the minute. The patient's condition improved immediately after the injection of vaccine and the administration of an ordinary bronchitis mixture. The following week the child contracted colitis from the baby next door, but even then she did not vomit and her cough became steadily less.

Neither pneumonia nor bronchitis developed in any of my patients after the commencement of treatment with the pertussis vaccine. I am sure it is a valuable remedy for this most distressing of infectious ailments in children.

Reviews.

THE TEETH OF THE BANTUS.

A GREAT deal of work is being done in various parts of the world in an attempt to complete a preliminary physical anthropological survey of the various races of mankind. Among the more important parts of this work is the task of recording accurate data with regard to the teeth. As Sir Arthur Keith points out in the foreword to Dr. Shaw's book on the teeth, "teeth are key structures, providing clues to race and to bodily health."[1] Also, on account of their more resistant qualities, teeth are the parts of fossil remains which are most likely to be preserved, so that a comprehensive knowledge of their structure is essential in order to base conclusions with regard to the probable identity of fossil remains.

Several important contributions have been made to our knowledge of the teeth and jaws of certain races, and Dr. Shaw has based his work to some extent on the excellent model provided by Dr. T. D. Campbell, who has given us detailed measurements and observations concerning the teeth and jaws of the aborigines of Australia.

The present contribution consists of a detailed description of the teeth and adjacent parts of Bantu races of South Africa, as well as a description of the customs of tooth extraction and mutilation in connexion with certain ethnic ceremonial rites in African races generally.

Other topics dealt with are developmental abnormalities, functional and pathological conditions, and the eruption of teeth.

Among his conclusions the author shows that while the teeth of the Bantu are smaller than those of the Australian aboriginal, they are larger than those of the modern European or bushman. The upper incisor teeth present the three "primary or primitive" forms of crowns described by Leon Williams. The morphology as well as the size of the Bantu teeth is, on the whole, uniform and true to type, although cases are recorded of diminutive and atypical teeth. Absence of third molars occurs with relative frequency, while examples of supplementary teeth are also met with. Mal-occlusion is apparently exceedingly uncommon, and the Bantu are largely immune to dental caries.

The tables of measurements are very complete, the illustrations are excellent and well reproduced, and on the whole, the work is a very valuable contribution to physical anthropology and reflects great credit on the author.

[1] "The Teeth, the Bony Palate and the Mandible in Bantu Races of South Africa," by J. C. M. Shaw, M.A., B.Dent.Sc., D.D.S., F.I.C.D.; with a foreword by Sir Arthur Keith; 1931. London: John Bale, Sons and Danielsson Limited. Demy 4to., pp. 150, with illustrations. Price: 12s. 6d. net.

Notes on Books, Current Journals and New Appliances.

A STORY OF EXPLORATION.

"LASSETER'S LAST RIDE," by Ion L. Idriess, is a vivid description of the trials and great tribulations of the quest expedition sent out to discover a gold field "rich and rare," and financed by the Central Australian Gold Exploration Company.[1] Mr. Idriess, who spent some years wandering with the tribes of Central Australia, has added a poignant page to the great Australian saga. His wealth of detail and intimate knowledge of tribal customs make this record of Lasseter's strenuous endeavour to relocate his El Dorado an epic. Even with an equipment which included an aeroplane, the expedition suffered innumerable hardships, accidents and disappointments. Menaced by unfriendly natives, haunted by the constant need of fresh water supplies, and finally forced by circumstance to separate from his companions, Lasseter refused to give in and battled on alone with a courage which is inspiring. Dying, blinded and defeated in his ambition, he left enough directions for a new expedition to be planned. Mr. Idriess has pieced the story together from the records left by the ill-fated explorer whose courage and fortitude rank second to none with the early Australian history makers.

PICTURES OF AUSTRALIA.

"THE FIFTH CONTINENT" is a book of reproductions of the finest photographs of Australia that we have seen.[2] Mr. Hoppé in this volume has sustained his reputation as an artist. Every aspect of Australian life, particularly life in the country, is depicted. The book is ideal as a gift to friends overseas.

[1] "Lasseter's Last Ride: An Epic of Central Australian Gold Discovery," by I. L. Idriess, with foreword by H. Basedow, M.A., M.D., B.Sc.; 1931. Australia: Angus and Robertson. Crown 8vo., pp. 241, with illustrations. Price: 6s. net.

[2] "The Fifth Continent," by E. O. Hoppé; 1931. London: Simpkin Marshall; Australia: Angus and Robertson. Royal 4to., pp. 192, with photographic plates. Price: 21s. net.

The Medical Journal of Australia

SATURDAY, NOVEMBER 21, 1931.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

STERILIZATION OF THE UNFIT.

FROM time to time men in public life and writers in the daily Press raise the question of sterilization of the unfit as a means of protecting society from the depredations of the mentally defective, of preventing increase in their numbers and of reducing the enormous and steadily increasing financial burden imposed by them on the State. There is no need in a journal such as this to refer to the great army of mental defectives in the community; to their association with habitual drunkenness, prostitution, venereal disease and crime; to their prolificity; nor to the economic aspect. Medical practice brings its devotees into a contact, at once so intimate and discerning, with people of all types, that they cannot fail to know these things. Medical practitioners cherish the principles of preventive medicine and would see them carried into practice. But not all those who clamour for sterilization of the mentally defective, are serious students of preventive medicine or of social problems in any shape or form. Many of them are students of nothing. Some of them (even perhaps medical practitioners among their number—who knows?) are seekers for notoriety. Some are cranks. Some may be defective themselves. In these circumstances there is danger that sterilization may be made a political catch-cry and that hasty and ill-considered legislation may be enacted.

The causes of mental deficiency may be included under three headings: those resulting from birth injury, those resulting from disease such as encephalitis and epilepsy and from trauma, and those due to heredity. Sterilization may be undertaken either in order to deter the individual from antisocial acts or to prevent him from reproducing his kind. Antisocial acts of a certain order may be rendered impossible only by asexualization, castration. It would be well if surgical operations of this kind were considered in a category by themselves. They would probably be undertaken after commission of a criminal act and in many instances would undoubtedly act as a preventive against the repetition of such acts. Sterilization, of course, can be effected without castration, and such a procedure would be undertaken soon after puberty to eliminate the possibility of a defective progeny. Not all the mental defects arising from the conditions enumerated and from similar types of condition will be transmitted to progeny, and this must continually be borne in mind. For example, a cerebral hæmorrhage that is the result of birth injury in an otherwise healthy baby, may cause a pronounced mental defect in the individual, but it is not at all likely that such a defect would be transmitted to the individual's children. It is also necessary to remember that the operation of sterilization is not without a certain, though small, risk to the patient. In the male, vasectomy is a minor surgical procedure and practically devoid of risk. Sterilization in the female calls for an abdominal section and, according to some observers, cannot be regarded as *un fait accompli* unless the intramural part of the Fallopian tube is excised with a wedge of tissue from the cornu of the uterus. These considerations raise the question as to how far sterilization, if it were enacted, should be made compulsory. Perhaps the point which is lost sight of by most non-scientific advocates of sterilization, is that the laws of heredity are not simple and that it would never be possible by any form of compulsory sterilization to eliminate defectives from a community. Opponents of sterilization, on the other hand, refuse to realize that sterilization, judiciously carried out,

would have a considerable effect in improving the mental and physical standard of the community. In summary we may conclude that mental defect is often inherited, that mental defectives are prolific, that sterilization is a simple procedure in the male and practically free from risk in the female, that sterilization does not affect the individual except in regard to his ability to procreate, that sterilization of minors must be legalized before it can be undertaken for eugenic reasons alone, and that the same holds true of compulsory sterilization in adults—in short, that some form of enactment to permit sterilization of mental defectives is urgently needed.

Having arrived at this conclusion, we must go farther. In several countries voluntary sterilization of minors is permitted, and this should be the aim, at any rate at first, of legislation in Australia on this matter. (Castration for definite offences is not here included.) At first there might be some difficulty in persuading parents and guardians of defective children to permit sterilization; but in time opposition would doubtless become less. A start might be made with defectives of the most pronounced types. If we turn to adults, it is obvious that if the patient and everyone concerned with his possible offspring consent, surgical operation can be carried out. There are, however, circumstances in which legislation is called for. As far as psychotic patients are concerned, the matter is urgent. It has been estimated that at the present time about 20% of the patients admitted to mental hospitals have been patients in such institutions on a previous occasion. This means that when a patient has recovered to a certain point and is allowed out "on trial," he is given *carte blanche* to bring into the world other individuals who may inherit a predisposition to the mental condition from which he is suffering. In any enactment dealing with this subject provision should be made for the sterilization of patients with mental disease as a condition to their discharge from hospital, whether the discharge is temporary or likely to be permanent.

There remains a final statement. Much of the mental defect in any community is the result of preventible disease and unhealthy environment. To attempt the reduction of mental defect by sterilization while preventible causes are allowed to operate is illogical and gives a false sense of security. If sterilization is to be considered at all, ministers of health must take care that the principles of preventive medicine and social hygiene are being practised in their entirety. Though much remains to be done in both these spheres, a great deal has been accomplished; the remainder can with a little energy be put in train. Sterilization of the unfit should be considered and it should be fathered by those who understand its possibilities and its limitations, and not by the ignorant, the notoriety-seekers or the cranks. National councils of mental hygiene and similar bodies exist; they have large committees of thoughtful people. Here is a field for the expenditure of their energy which at present appears to be latent.

Current Comment.

TUBERCULOUS ENTEROCOLITIS.

THE phthisical patient's hope of cure depends largely on his ability to absorb a sufficient quantity of suitable nourishment. It is important that his digestive organs should be in good order; if he is subject to gastro-intestinal disturbances, his powers of absorption are reduced and the problems of his treatment are correspondingly increased. It is well known that the phthisical patient is apt to suffer from digestive upsets and diarrhœa, and that tuberculous enterocolitis may be a complication of pulmonary tuberculosis. Hypochlorhydria and achlorhydria are common. In the opinion of many, insufficiency of the gastric hydrochloric acid is the cause of diarrhœa in the majority of instances, and enterocolitis is a comparatively rare development. Others hold that enterocolitis is the commonest cause of intestinal disturbances. The matter requires elucidation. It is remarkable that, while much investigation has been made into the treatment of tuberculous lesions of the lungs, and some elaborate therapeutic procedures have been evolved, comparatively little attention has been paid to the gastro-intestinal tract, on whose good function the patient depends after all for his very existence. A report by Andrew L. Banyai may prove to be an important contribution on the subject.[1] The value of the introduction of air into the peritoneal cavity, in the treatment of tuberculous peritonitis, has long been recognized; it is only recently that similar methods have been employed in the treatment of enteritis; Banyai believes these methods to be of considerable value.

[1] *The American Journal of the Medical Sciences*, September, 1931.

He remarks that intestinal tuberculosis is a far more frequent complication of pulmonary tuberculosis than is generally believed. In as high a proportion as 92·6%, persons that have died of pulmonary tuberculosis, have been found to be affected with tuberculous ulceration of the intestines. Thus it becomes clear that most persons suffering from advanced pulmonary tuberculosis are subject to ulcerative enterocolitis. But ulceration, hyperplasia and cicatricial stenosis do not occur until late in the course of the disease. In the earlier stages the lesions consist of tubercles in the mucosa or the lymphatic tissue of the submucosa; they may persist for long periods without ulceration. Symptoms vary widely, and their severity is not in direct proportion to the extent or severity of the lesions. Anorexia, a sense of undue fullness after eating, nausea, constipation, abdominal pain relieved by fasting and aggravated by eating, *et cetera*, are among the symptoms of early enterocolitis. At the same time there are likely to occur constitutional disturbances that cannot be accounted for by the condition of the lungs. Diarrhœa, he declares, is usually indicative of extensive involvement. He admits that the absorption of toxins from active tuberculous foci in the lungs may cause hypochlorhydria and resulting gastro-intestinal disturbances. He also states that if symptoms are not relieved by an improvement in the pulmonary condition, search should be undertaken for tuberculosis of the intestines. It is important that an early diagnosis should be made. Though he states that it "is possible to establish the diagnosis of intestinal tuberculosis on the basis of symptoms and physical findings," he is careful to remark that, even when radiological and bacteriological data are also available, a correct diagnosis may be impracticable; an added difficulty is that in some instances extensive ulceration gives rise to no suggestive symptoms. He points out that the prognosis of intestinal tuberculosis is not necessarily hopeless; for in animal experiments complete healing of ulcerative and exudative lesions has been observed. He might have added that healing is known to occur in tuberculous ulcers of the human intestine.

Treatment may be prophylactic or therapeutic. Kantor suggests four measures for use in prophylaxis: the prohibition of swallowing tuberculous sputum; the administration, when necessary, of hydrochloric acid; the prevention of intestinal stasis; the careful investigation of every tuberculous dyspeptic. Therapeutic treatment may consist of dietetic measures, the administration of medicines, X ray or ultra-violet irradiation, surgical measures, or the establishment of artificial pneumoperitoneum. Food should not be forced beyond the patient's tolerance; small quantities of bland food, free of cellulose, should be given; nothing that is likely to ferment should be administered. When diarrhœa is pronounced, the diet should consist of liquids only. But in addition to dietetic measures other procedures must be undertaken.

Banyai injected oxygen into the peritoneal cavity of forty-four patients who had failed to respond satisfactorily to dietetic and medicinal treatment. The method of administration is simple. The patient evacuates the bladder. No enema or aperient is given. An adequate dose of codeine is administered one hour before the operation, if the patient is subject to severe coughing. A point in the lower left quadrant of the anterior abdominal wall is locally anæsthetized, and the needle is passed obliquely upwards and inwards. If the needle is passed directly, there may not be a satisfactory closure of the wound and gas may leak out. Usually the intraabdominal pressure of untreated patients is equal to atmospheric pressure; in rare instances in Banyai's series there was a positive pressure of 0·5 to 1·0 centimetre of water. A positive pressure usually resulted after the introduction of 300 to 500 cubic centimetres of oxygen, but sometimes not until 1,000 cubic centimetres had been given. Thus the manometer reading is not such a useful guide as it is in the performance of artificial pneumothorax. The disappearance of liver dulness and the feelings of the patient give more reliable information. Other interesting observations on the intraabdominal pressure were made; for these the reader is referred to the original paper. The greatest quantity of oxygen administered at one sitting was 1,500 cubic centimetres, the smallest 150 cubic centimetres. Patients were apt to experience a certain degree of discomfort due to tension or the occurrence of pains in the shoulders; this was quickly relieved as a rule by raising the foot of the bed. As a result of this measure the gas shifts away from the diaphragm towards the pelvis. Inflation is repeated at intervals varying usually from one to two weeks.

Relief from symptoms resulted in thirty-one cases; there was no relief in thirteen. Banyai remarks that fourteen patients were completely relieved, but he does not make it clear whether they were completely relieved of subjective symptoms or apparently cured of the disease. He suggests that the good effects of oxygen administration may be due either to an increase in the cell activity of the body and consequently a more efficient immunological response, or to local irritation and hyperæmia.

Perhaps it is open to argument whether or not all Banyai's patients were affected with tuberculous enterocolitis—the difficulties in the way of diagnosis are numerous and great. But they certainly suffered from distressing gastro-intestinal disturbances, from which they were relieved in many instances by his treatment. The treatment therefore appears to be justifiable, if only because it promises a good function of the alimentary tract. The mechanism by which the improvement is effected can only be guessed at; further investigation is necessary. Observations on the intraabdominal pressure are of additional interest on account of the view held by some that a negative pressure is concerned in the causation of visceroptosis.

Abstracts from Current Medical Literature.

THERAPEUTICS.

Hypnotics.

G. P. GRABFIELD (*The Journal of the American Medical Association*, May 30, 1931) gives some observations on the value of commonly used hypnotics. A number of these drugs were given in a series of cases. Half the pharmacopœial dose or half the dose recommended by the manufacturers was administered and repeated at intervals until a good night's sleep was obtained. Some twenty or more patients were tested with each drug and the results tabulated. It was found that chloral hydrate was the most efficient, and barbital, as originally introduced in 1903, was second in efficiency. These two drugs were also much less expensive than others. It is suggested, therefore that chloral hydrate and barbital should be used for simple insomnia. A small dose was often found as efficient as larger doses.

Use of Calcium in Peristaltic Pain.

W. BAUER, W. T. SALTER AND J. C. AUB (*The Journal of the American Medical Association*, April 11, 1931) discuss the administration of calcium in lead colic and give the results of experimental intravenous injection of calcium chloride in lead colic, ureteral colic and hepatic colic. There is an analogy between the metabolism of calcium and lead. The administration of various decalcifying agents results in an increased excretion of lead and forms of therapy promoting the retention of calcium favour the retention of lead. In the treatment of lead poisoning a high calcium diet is employed, and a complete subsidence of symptoms usually occurs in twenty-four to forty-eight hours. With a view to hastening calcium retention and obtaining more prompt relief from severe colic and other symptoms of lead poisoning, calcium chloride was injected intravenously. A sterile 5% solution was used in amounts varying from five to twenty cubic centimetres. To insure the minimum of untoward symptoms it was always given at the rate of two cubic centimetres a minute. During the administration the patient complains of an intense feeling of heat over the entire body, burning of the tongue, and occasionally is nauseated and vomits. There is a fall in the blood pressure of from 10 to 40 millimetres of mercury and a prolonged elevation of the skin temperature of from 1° to 3° C. Escape of the solution into the surrounding tissues will cause sloughing. Injection can be repeated in four hours. The results in the treatment of twenty-four patients suffering from lead colic were dramatic. There was immediate cessation of pain frequently before the injection was completed. In some patients the pain recurred several hours later, but could always be relieved by a second injection. Two patients, who were only temporarily relieved, were found to be suffering from a perforating type of duodenal ulceration. Failure should always raise the suspicion of some other condition than lead poisoning. Equally prompt relief was obtained in twelve patients with ureteral colic, in one of whom morphine had had no effect. In five out of six patients suffering from hepatic colic there was similar prompt relief. The one patient unrelieved was found at operation to be suffering from chronic gall-bladder disease without stones. The almost instantaneous relief of lead colic following the intravenous use of calcium chloride is not to be explained on the basis of suddenly increased skeletal storage of calcium and lead. The therapeutic benefit derived in other types of colic enables calcium to be classified as an antispasmodic. Its beneficial use in the various types of tetany has long been appreciated. In such patients the spasmodic contractions are due to calcium deficiency.

Urinary Acidifiers.

V. E. HENDERSON AND J. M. SCOTT (*Canadian Medical Association Journal*, June, 1931) discuss urinary acidifiers and alkalinizers. Hexamine acts only in acid urine; acid sodium phosphate with *syrupus limonis* is best given separately from hexamine; this will have a slight effect in acidifying the urine if given four times a day. A mixture of ammonium benzoate, 1·2 grammes (twenty grains), liquid extract of liquorice, 1·0 mil (fifteen minims), and syrup, 4·0 mils (one fluid drachm), with half an ounce of water, and diluted four to eight times with water to cover the disagreeable taste, is more efficacious as an acidifier. Ammonium chloride is more useful still, and can be given with hexamine, 1·2 grammes (twenty grains) of each, with liquid extract of liquorice, 0·6 mil (ten minims), and syrup, 4·0 mils (one fluid drachm). If there is pain due to the acid urine, acid boric and potassium citrate, 1·2 grammes (twenty grains) of each, may be given, or 2·4 grammes (forty grains) of potassium citrate with 2·4 mils (forty minims) of tincture of hyoscyamus to relieve spasm.

Ergosterol and Parathyreoid.

N. B. TAYLOR, C. B. WELD, H. D. BRANION AND H. D. KAY (*Canadian Medical Association Journal*, June, 1931) describe experiments in dogs and other animals in which the parathyreoid glands had been removed. Infantile tetany is relieved by parathormone or by cod liver oil or ergosterol (irradiated); it is due to vitamin *D* deficiency. The parathyreoid glands were removed from eight dogs, the serum calcium was reduced as a result, and the dogs went into severe tetany. One cubic centimetre of irradiated ergosterol by mouth immediately relieved the symptoms; the dose was repeated after twenty-four hours, and the animals remained free from tetany for one or two weeks. It appears that ergosterol is as effective as parathormone in relieving this condition in dogs. In other animals all accessory parathyreoid tissue was removed in addition to the thyreoid and associated parathyreoids; the tetany which developed, was not relieved by large doses of irradiated ergosterol (one or two cubic centimetres twice daily for several days). Overdosage with irradiated ergosterol was studied in forty dogs, one cubic centimetre being given twice a day; the serum calcium rose considerably (19 milligrammes per 100 cubic centimetres). The dogs became depressed, weak, lethargic, atonic and emaciated; vomiting, diarrhœa and bloody stools preceded collapse and death in a few days. At autopsy a hæmorrhagic state of the gastro-intestinal tract was noted as far as the lower part of the ileum, and the bone marrow was definitely hæmorrhagic. These conditions were exactly similar to those occurring due to parathormone overdosage. The inorganic phosphorus content of the whole blood was found to show an early fall and a late high level (20 milligrammes per 100 cubic centimetres) after the administration of excessive doses of ergosterol, similar to the effect after large doses of parathormone. Similar marked increase of the non-protein nitrogen (up to 200 milligrammes per 100 cubic centimetres) was observed with both these substances. Hunter, Aub and others have shown that the excess serum calcium in these conditions is derived from the cancellous tissue of the bones. Parathormone causes increased blood calcium and lead concentration in the blood in lead poisoning and increased excretion of these substances in the urine. It has been shown that in lead poisoning the metal is deposited in the bones in the form of tertiary lead phosphate. Similar results occur when irradiated ergosterol is given to cats overdosed with lead acetate. Ergosterol will certainly raise the serum calcium of animals on a normal or excessive calcium-containing diet. It follows that too much calcium may be excreted when excessive doses of ergosterol are given.

Treatment of Muscular Rheumatism.

D. NATAUNSEN (*Deutsche Medizinische Wochenschrift*, May 22, 1931) discusses the various lines of treatment for muscular rheumatism. He prefers infiltration of the surrounding tissues with hypertonic glucose solution. The pain is generally due to stimulation of the peripheral nerves, and this in its turn causes hypertonus of the affected muscles. The vicious circle is completed by further stimulation of the nerves by the muscles. An infiltration of the affected area with a 10% glucose solution in amounts varying from 50 to 100 cubic centimetres results in immediate relief of pain. Similar results have followed its use for intercostal neuralgia, facial neuralgia and sciatica, although the quickness and permanence of relief is not quite so pronounced as with muscular affec-

tions. Failure should always raise the suspicion that arthritis is the cause of the pain. The only contraindication is the presence of diabetes.

Treatment of Migraine.

J. KLAUSNER-CRONHEIM (*Deutsche Medizinische Wochenschrift*, August 21, 1931), being struck with the fact that attacks of migraine generally disappear during pregnancy, considered that this may be due to the increase in anterior pituitary hormone then circulating in the blood. Accordingly she administered the anterior pituitary hormone to a series of ten patients with migraine. Tablets of "Prolan," each containing 450 mouse units, were given by mouth thrice daily. In three cases no success was noted, but the attacks in the other seven either ceased or became much milder and less frequent. All the patients had been under treatment of various sorts for a considerable time. While she does not claim this treatment to be specific in all cases, it promises, in her opinion, to be a valuable aid in treatment.

NEUROLOGY.

The Cerebral Cortex During Unconsciousness.

R. L. WORRALL (*Journal of Neurology and Psychopathology*, April, 1931) writes a critical review of the theory of conditioned reflexes, with reference to the symptoms of epilepsy and narcolepsy. He quotes freely from the work of Pavlov and Kinnier Wilson. His conclusion is that experimental and clinical evidence supports the view that unconsciousness is the result of a process of physiological inhibition extending over the whole of the cerebral cortex. This irradiation of cortical inhibition spreads from a focal point of initial stimulation. In epilepsy the focal point of stimulation is in the motor area of the cortex—the area of stimulation may include the whole motor cortex, with, in cases of unconsciousness, an irradiation of inhibition over the rest of the cortex through reciprocal induction. In many cases of narcolepsy the point of initial stimulation is also in the motor cortex, but is distinguished by being the coordinating point for efferent impulses to the facio-respiratory muscles, *viâ* a separate path, causing "involuntary" expression of emotion. The following quotation from Kinnier Wilson ends the review: "Inhibition of tone, inhibition of innervation—local or general, cortical or infracortical, or both—here surely is the clue to the diverse phenomena of narcolepsy, cataplexy and probably also of catalepsy, so intimately interrelated.

Intramedullary Gliomata of the Spinal Cord.

H. CAIRNS AND G. RIDDOCH (*Brain*, June, 1931) report two cases to show that certain intramedullary gliomata can be removed without serious permanent injury to the spinal cord. In one case the tumour extended from the fourth cervical to the third thoracic segments, in the other from the third to the fifth thoracic segments. Preoperative evidence that a spinal growth is within the substance of the cord is always inconclusive, but signs indicating a lesion of considerable length in combination with dissociated sensory loss (analgesia dominating anæsthesia) in the areas supplied by the affected segments may be taken as guides. Hitherto, in the operative treatment of such growths it has been usual to rely on the so-called extrusion method; in these two cases, however, after the growths had been exposed by a posterior longitudinal incision, it was clear that they would not extrude themselves; accordingly they were removed by gentle dissection. Considering that the tumours were 11·0 centimetres and 4·5 centimetres long respectively, and had greatly canalized the cord, the subsequent recovery of motor, sensory and reflex function was extraordinary.

Histological Study of Encephalitis.

ROBERT CARMICHAEL (*Journal of Neurology and Psychopathology*, January, 1931) describes the findings in four cases of chronic epidemic encephalitis. The lesions were diffusely and extensively distributed throughout the nervous system. Not only was the *locus niger* the seat of important changes, but such were found in all basal nuclei and in the cerebral cortex, and in some cases in the spinal cord. The morbid process was thought to be an active one; the lesions were in an evolutionary stage, not cicatrices. They exhibited destructive characters, degenerated cells were habitually surrounded by abnormal formations, there were indications of diapedesis in the neighbourhood of small vessels, and large astrocytes pushed their processes into degenerating nerve cells. The longer the disease had lasted, so much the more were the nerve elements affected; in more rapid cases perivascular lesions dominated the picture. It would appear that the long maintenance of activity in these processes was conditioned by tardy but sustained defence reactions.

The Menopause and Psychosis.

C. B. FARRAR AND R. M. FRANKS (*American Journal of Psychiatry*, May, 1931) hold that most women who show insanity at the menopause have been potentially psychotic all along. The exact relation between mental disorder and the endocrine and other changes attributed to the menopause is unknown and the age factor in most cases is inconspicuous. It is admitted only that since the climacteric is a critical period, not only biologically, but often in the life relations of the individual, the psychosis may be reactive to such external conditions. On the whole the morbid effect of the climacterium is exaggerated. There is a widespread assumption that trouble may be looked for when the change of life comes, just as many people expect to be seasick when they cross the ocean. This state of the feminine mind is unfortunately encouraged all too often by the medical profession, and the opinion is too readily expressed that difficulties of all sorts arising during the rather wide span of the transition years are to be attributed to the menopause. While the menopausal psychoses as a unitary classification have disappeared from text books, the dread of them remains, and this it is the duty of the profession to counteract.

Korsakoff's Syndrome.

E. ARNOLD CARMICHAEL AND RUBY O. STERN (*Brain*, June, 1931) state that confusion exists regarding Korsakoff's syndrome, and that if the term is to be used, it should be applied to the symptom complex which Korsakoff observed, and not, as is sometimes done, to the attendant psychical phenomena alone. What Korsakoff observed was a peculiar psychical state characterized by amnesia, disorientation in time and space, and a tendency to fabrication, accompanied by signs of peripheral neuritis. This peripheral neuritis, while commonly due to alcohol, may have other toxic cause. The writers report five cases, all of which happened to be associated with alcoholism and which came to autopsy. Constant histological findings in the cerebral cortex were excessive deposits of lipochrome in all nerve cells and in other elements. They view the changes as specific and as indicating a "central neuritis." They also comment on the fact that similar changes have been found in pellagra and accordingly suggest that in both Korsakoff's syndrome and pellagra the highly specialized nerve cells of the cortex lack some essential constituent which ordinarily fortifies against attack and damage by toxins.

Epilepsy and Gun Shot Wounds.

W. E. STEVENSON (*Brain*, June, 1931) states that a survey of official documents at the Ministry of Pensions (Great Britain) in 17,300 cases of head wound shows that epilepsy has been recorded as a secondary disability in only 270 cases. In most this epilepsy has arisen within a few months of injury, but there have been authentic instances in which the interval has extended to more than ten years. Of course, when there has been a long latent period with freedom from symptoms, other possible cause than trauma has been considered. Epilepsy following superficial wounds of the head may often have been psychogenetic, the outcome of shock or fear acting upon an unstable nervous system, and such epilepsy has followed rapidly upon the injury. The fits in essential traumatic epilepsy have been on the whole indistinguishable from those of idiopathic epilepsy. But vertigo, "functional" attacks marked by loss of emotional control, and epileptic equivalents have been more frequent as complications. Also there has been more mental deterioration and this in proportion to the severity of the wound and the number of the fits. True Jacksonian epilepsy has been rare.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE QUEENSLAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the B.M.A. Building, Adelaide Street, Brisbane, on July 3, 1931, DR. F. A. HOPE MICHÔD, the President, in the chair.

The Diagnosis of Acute Appendicitis.

DR. ALAN E. LEE read a paper entitled: "Acute Appendicitis: Its Early Diagnosis" (see page 635).

DR. M. GEANEY thanked Dr. Lee for his very important paper on a condition which was very common and was so important in surgery. With regard to the figures showing the increased death rate in the United States of America, it was alarming to think that there were so many more deaths from the disease than formerly. He considered that this increase was probably due largely to wrong diagnosis, though medical men were not any less likely to diagnose appendicitis now than then. Of course, appendicitis might be a more prevalent disease now than thirty years ago. With regard to the types of appendicitis, in the condition of acute inflammation of the appendix and in those cases in which there was no obstruction, the condition was not really a surgical one, but when further inflammation of the appendix occurred, the appendix was liable to perforate, and Dr. Geaney therefore thought that any acute inflammation of the appendiceal wall was a surgical condition. The patients became so used to having attacks of appendicitis that they diagnosed the condition themselves, left it too long, and the appendix was perforated when operation was performed. Also, once the wall of the appendix was damaged, being a redundant organ, it was liable to set up other conditions, for example, gall-bladder disease, peptic ulcer *et cetera*. Once it was damaged, therefore, the appendix was better removed.

Leucocytosis was occasionally important in differentiating appendicitis from other diseases. As a house surgeon Dr. Geaney had carried out a leucocyte count on many cases of appendicitis, and he had never seen one of those cases of acute appendicitis operated on that did not have a good leucocytosis. Pyuria was another condition often found to confuse the diagnosis, and operation on a case of acute appendicitis might be deferred on account of it. A carefully taken history was valuable, for it would show that the pyuria did not mean a primary kidney or bladder condition. Dr. Geaney's experience was that the amount of pus in the urine due to irritation of the renal tract by an acutely inflamed appendix was much less than that of a primary kidney or bladder inflammation with the same constitutional disturbance. A low temperature was most commonly found in acute appendicitis, but occasionally the temperature would be raised to 104° F. Another condition resembling acute appendiceal obstruction, in which on operation the appendix looked very innocent, was threadworms. One did occasionally find patients who complained first of pain in the right iliac fossa, and, however carefully one took the history, one could not get them to admit upper abdominal pain or discomfort, and so one had to rely on physical signs sometimes.

John D. Murphy in 1910 said that the cardinal symptoms of appendicitis were: pain in the abdomen, sudden and severe, often referred to the upper part of the abdomen; nausea and vomiting three to four hours after the pain (though Dr. Geaney considered it was sooner); elevation of temperature two to twenty-four hours after onset; leucocytosis. If the nausea and vomiting preceded the pain, the condition was not one of appendicitis.

In contradiction to this, Dr. Geaney said he remembered one case in which the nausea preceded the pain by a couple of hours. A rise of temperature was never absent at some time in the first thirty-six hours. He had never found a leucopenia.

Sargent in 1912 stated that the symptoms were sudden in onset and well marked, or else indefinite malaise, indigestion and constipation might precede them. Dr. Geaney agreed with Dr. Lee that the history was the main thing in the examination of the patient as to whether the patient had an acute appendicitis, but the physical signs should not be neglected.

DR. HEDLEY BROWN thanked Dr. Lee for his instructive paper. Dr. Brown showed a specimen of an appendix, removed from a woman sixty-six years of age, who had had pain in the right side of the abdomen for twelve hours. Vomiting was present, the temperature was not raised, and the pain began in the right side and remained there. The urine contained albumin and pus. Operation was performed and the appendix removed. One inch of the appendix at the base was found to be normal; the rest was obstructed and gangrenous. This patient had denied any epigastric pain. In contrast was a second patient, a man twenty-nine years of age, who had given a typical history of appendicitis. The pain had begun in the epigastrium, had been round the umbilicus and had then centred on the right iliac region. This patient was found to have a lymphosarcoma in the ilium, and this was quite consistent with a mid-gut lesion.

DR. DONALD CAMERON thanked Dr. Lee for his interesting paper. One aspect of the disease had not been touched on, and that was the question of appendicitis in children. Dr. Cameron remarked on the difficulty of getting a history from parents, and said one had to make a diagnosis on physical signs in the majority of cases. Threadworms in the appendix could cause symptoms of an acute appendicitis, and Dr. Cameron had frequently seen this in a child who seemed very ill with a high temperature, and in whom operation revealed an appendix full of worms. Another condition which had not been mentioned, was appendicular colic, in which a practically normal appendix was found. A pneumococcal condition of the abdomen could be mistaken for appendicitis and operation performed. This was particularly difficult to diagnose in children, pain round the navel being a constant sign. Pneumonia in children with referred pain in the abdomen also came into the differential diagnosis.

DR. E. S. MEYERS said that with his usual thoroughness Dr. Lee had gone into the whole matter. On the theoretical side perhaps one could not agree absolutely with him. The subject could be looked at in two ways: from the point of view of the general practitioner and from the point of view of the hospital surgeon. The general practitioner would go out, in the night frequently, at the beginning of the attack, and at that stage could not be sure what was happening. He would perhaps see the patient two or three times before he decided. If the patient were followed over one day the syndrome described by Murphy was valuable and worked out in the majority of cases. When the general practitioner was called in to an attack which had lasted some days, different things came into the differential diagnosis. Murphy mentioned two conditions that could be confused with it, a kidney condition, such as a calculus, and gall-stones, and Dr. Meyers had found these two the most difficult to differentiate. A chronically inflamed appendix showed many difficulties, for example, with the question of a new growth.

From the point of view of the hospital surgeon there were several factors to account for the high mortality, for example, salt medication; medication by the chemist; doses of oil to shift the pain which, instead, shifted the wall of the appendix; the young resident medical officer who considered the man outside probably did not know much about it; and the honorary surgeon who was liable to make mistakes.

Dr. Lee in reply said that he was very gratified with the reception accorded his paper. A more correct title might have been "The Diagnosis of Early Appendicitis." When mentioning operation for appendicitis, he wished to stress that he meant emergency operation. Many of the cases of appendicitis which he classed as medical, would certainly have to be operated on at some future time. In spite of criticism, Dr. Lee still thought it true, in the majority of cases, that an obstructive appendicitis was the only type that exposed the patient to danger to his life, and that this type almost always gave a history of epigastric pain.

With regard to leucocytosis, Miller had dealt fully with this and did not find it of any diagnostic value.

Not only obstruction, but also irritation in the mid-gut loop could produce intrinsic epigastric pain. This explained those cases of appendiceal colic in which threadworms were the causal agent. In an adult Dr. Lee had recently observed epigastric pain associated with severe diarrhœa; in this instance a small splinter of wood and some hair were found in the appendiceal lumen, and evidently were the causal factor in the production of symptoms.

In reply to Dr. Hedley Brown, Dr. Lee said the specimen shown was an example of an appendix buried in its mesentery. The mesentery carried up with it a thin layer of the sensitive subperitoneal fascia, and such an appendix behaved exactly as a retroperitoneal appendix might behave. He had pointed out that with a retroperitoneal appendix the parietal pain was so early and so acute that the intrinsic epigastric pain sometimes appeared to be blotted out. Lymphosarcoma explained itself as an example of mid-gut loop obstruction.

He could not speak with much experience of appendicitis in children, but the high mortality associated with the early years of life emphasized the disadvantage of having to wait for physical signs rather than having to depend on the history for guidance. He reiterated that accuracy of diagnosis in acute appendicitis could be increased if the necessity of epigastric pain were made the working rule before an emergency operation was carried out.

A MEETING OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at Warragul on July 18, 1931.

Achlorhydric Dyspepsia.

DR. J. MOORE ANDREW read a paper entitled: "Achlorhydric Dyspepsia" (see page 644).

DR. C. F. MACDONALD, on being asked his opinion of the skiagram in the case reported, said that he had examined the films and did not think that one could class the case amongst those in which the X ray findings had been definitely misleading. The case really opened up the question of the economics of X ray work. It was necessary to remember that three things were required: first, repeated screenings; second, many films; and third, large and powerful apparatus to enable a fast exposure to eliminate the effect of peristalsis.

DR. J. R. BELL congratulated Dr. Andrew on his informative paper, which indicated much care and thoroughness in its compilation. He endorsed the plea for wider use of the fractional method of gastric analysis, and had for many years urged its value in the investigation of dyspepsias met with in general practice.

Achlorhydria was a very interesting subject, and he was glad to hear Dr. Andrew clearly distinguish between the terms achlorhydria and achylia. These terms were not synonymous, *achylia gastrica* being first used by Einhorn, of New York, to denote the complete absence of free hydrochloric acid and pepsin from the gastric secretion, while achlorhydria merely meant the complete absence of acid. In his experience achylia was very rare; in a series of 286 cases in which the presence or absence of pepsin was noted, he found it to be absent in only 8% of the cases. Achlorhydria was found in approximately 13% of all gastric analyses (537 of 4,091 private cases so investigated).

In order to avoid finding achlorhydria with misleading frequency, it was essential that the technique of the test meal be correct. This included the dexterous passing of of the tube, with a minimum of discomfort to the patient, the avoidance of undue swallowing of saliva, the oatmeal gruel meal to be appetizingly prepared and served, and representative specimens of the stomach contents withdrawn. It should scarcely be necessary to emphasize that the test meal be a fractional one and that the obsolete one-hour method should not be used.

The incidence of achlorhydria in the sexes was about equal; in a series of 491 cases 51·0% of patients were females and 48·9% were males. Women, however, were less likely to regain the lost hydrochloric acid, for in a series of cases in which achlorhydria was found to have persisted up to five years, 61·7% of patients were females and 38·2% males. Moreover, acid was less likely to return to the stomach if the patient was middle-aged or elderly, the average age of those in whom acid returned being 33·4 years, as compared with an average age of 44 years for those in whom the acid did not return, even up to five years after the first discovery of achlorhydria.

Achlorhydria was associated with many conditions, the order of frequency found by Dr. Bell in a series of 475 cases being: Functional dyspepsia, 35·1%; carcinoma of stomach, 12·6%; gall-bladder disease, 9·8%; chronic gastritis, 9·7%; gastrogenic diarrhœa, 5·9%; Addison's anæmia, 5·5%; gastric ulcer, 1·6%; phthisis, 1·5%; and migraine, 1·5%. It was of interest to note that achlorhydria was found in only 58·5% of 82 cases of carcinoma of the stomach, and if this condition was to be diagnosed before the advanced inoperable stage, one must not think that the presence of free hydrochloric acid rendered such a diagnosis improbable. To many it was still surprising to realize that achlorhydria was found in 10·3% of 68 proved cases of gastric ulcer.

Achlorhydria might be congenital, having been found by Bennett and Ryle in 4% of healthy medical students who had never had digestive disturbances. While it might be symptomless, it was more common to find such symptoms as described by Dr. Andrew. Diarrhœa frequently occurred, but constipation was commoner. In the characteristic gastrogenic type of diarrhœa hydrochloric acid therapy produced an immediate effect, even in small doses. It was still obscure why this type of diarrhœa was associated with achlorhydria so definitely, whereas constipation was a more frequent associate of achlorhydria. Possibly there was also a functional pancreatic achylia in these cases. Sometimes it was necessary to treat the irritability of the colon and secondary colitis that developed in long-standing cases of gastrogenic diarrhœa.

Asthenia was a common symptom of achlorhydria, and Dr. Bell had been impressed by the frequency with which patients volunteered the remark that they had regained their lost energy and strength after adequate hydrochloric acid therapy. He had seldom found a patient who could not take the necessary physiological dose of two drachms of *acidum hydrochloricum dilutum* (British Pharmacopœia) after each meal, provided the dose was gradually increased to this amount and due attention paid to such details as flavouring, sipping it slowly during and after the meal *et cetera*. Experimental work conducted at Guy's Hospital, London, had demonstrated that the maximum pharmacopœal dose of thirty minims was quite inadequate in restoring the free hydrochloric acid curve to normal. In conclusion, Dr. Bell showed the fractional test meal chart of a patient with achlorhydria who had sipped three drachms of dilute hydrochloric acid, diluted with twelve ounces of water, throughout the test, and the free hydrochloric acid curve obtained was absolutely normal. This patient had increased the dose of acid in error, but had continued to take it for almost a year owing to the benefit obtained therefrom.

DR. IVAN MAXWELL referred to the question of sepsis, for example, of the nasal sinuses. He had found in asthma patients that about 25% had absent hydrochloric acid; but almost all had pepsin in the stomach content.

Achlorhydria due to regurgitation of duodenal contents could be detected by estimating the total chlorides.

In regard to the patient who was taking three drachms of hydrochloric acid, there was the possibility of dilution with one-tenth normal sodium chloride secreted by the stomach.

DR. L. HURLEY congratulated Dr. Andrew. His own experience with achlorhydria had been disappointing. It was common in people over fifty; but there was no constant form of dyspepsia associated with it, and a large proportion of those with achlorhydria did not have any symptoms. He did not agree with Hurst that absence of hydrochloric acid was the cause of pernicious anæmia. As far as his experience of treatment with hydrochloric acid was concerned, the only type that had derived benefit,

were those cases associated with morning diarrhœa, and in these the dosage was inadequate to restore the acid to normal level. He found that many patients would not take the requisite doses, and even if they did, they did not derive benefit.

DR. D. G. THOMAS also congratulated Dr. Andrew on his excellent paper. He referred to a possible explanation of the discrepancy between X ray findings and test meal findings. It was obvious that when the stomach was examined by X rays, the most intensive study was immediately after the meal, and even when there might be some delay in the complete emptying of the stomach, the commencement of emptying might take place after a short interval and a fair percentage of the meal might leave the stomach while it was still under observation, giving rise to the idea of good motility. Serial films were necessary to demonstrate the emptying. As regards hydrochloric acid, they had to remember that it had a motility effect as well as its effect as an acid. He was glad to hear the advocation of test meal investigations.

DR. J. P. MAJOR congratulated Dr. Andrew on his very fine paper. He referred to the point raised by Dr. Hurley about the relation of achlorhydria to pernicious anæmia. It was a very interesting point, and he had doubts as to whether the absence of hydrochloric acid was an essential factor. It had been shown that pernicious anæmia could be cured by treatment with liver or gastric extract with or without hydrochloric acid.

DR. D. M. SILBERBERG referred to the fact that achlorhydria was associated with other conditions, such as rheumatoid arthritis and also other forms of anæmia and leuchæmia. The question was raised as to whether the loss of hydrochloric acid did not permit the entrance of streptococci, which might then act in different ways, for example, the hæmolytic forms giving rise to pernicious anæmia, others acting on the reticulo-endothelial system causing the leuchæmias, others acting on the joints giving rise to the types usually classified as rheumatoid arthritis.

DR. VICTOR HURLEY associated himself with the other speakers in congratulating Dr. Andrew. To his mind the chief value of the paper was that it represented original work. The cases were Dr. Andrew's own, the investigations were his own, and the paper had been compiled from his own experience, which made it very much more valuable than any paper simply compiled from the literature.

Dr. Andrew, in reply, wished to thank the various speakers for their very kind remarks. He would particularly like to refer to Dr. Bell, and was gratified to know that the results of Dr. Bell's analyses bore out his own limited experience.

In regard to Dr. L. Hurley, he considered in his own series he did get definite benefit from treatment with hydrochloric acid, although he had to admit there were a certain number who did not improve, particularly those in whom a nervous factor was important. One patient of his responded equally well to alkali or acid, and took either, according to his feelings. Possibly the variation in the amount of mucus caused the difference. He did think that there was evidence that the absence of hydrochloric acid was an important factor in pernicious anæmia.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Department of Physiology, University of Adelaide, on September 24, 1931. The meeting took the form of a demonstration by PROFESSOR C. E. HICKS and members of the staff of the department.

Teaching of Physiology.

In his opening remarks Professor Hicks stated that the demonstration consisted of three parts: one showing the mammalian physiology class in action; another, the human physiology class; and the third, some aspects of research at present in hand. He drew attention to the unorthodox nature of the course, emphasizing the object as being the establishment of a sound training in the principles underlying medicine and surgery. He stated that his own special training was responsible for this—he had been a clinical pathologist and was interested in disorders of function as found clinically, and had always felt that the orthodox training in physiology failed both to excite the interest of the medical student and to lay the emphasis on the points of value to him. Starting with Sherrington's decerebrate mammal course as a basis, he had developed a complete operative mammalian course covering two terms, followed by a course of human physiology, using man as the experimental object, for the remaining two terms. The mammalian course utilized the rabbit, which the students themselves anæsthetized by paraldehyde *per os* as a basal anæsthetic, supplemented by ether given by the open method. They performed all the operations themselves, studying cardiac, vasomotor and respiratory reflexes, diuresis, shock, hæmorrhage *et cetera*, and the pharmacology of these conditions. They operated in groups of five to a table, an anæsthetist, an operator and assistant, a technician, and an amanuensis; these rotated in function from day to day. Scrupulous discipline was observed, and the importance of team work was inculcated, the operator being in charge of a group during an experiment. It was claimed that not only was physiology better learned and more clearly understood from the actual performance of the experiment, but that the importance of care in handling living tissues, as well as scrupulous attention to all phases of function, was learned in the difficult art of keeping the animal alive during the operations. In this respect the rabbit was the best animal, since it was also the "worst operative risk." Ether anæsthesia was very prone to cause pulmonary œdema if not carefully administered to the rabbit, and hæmorrhage and shock were less readily recovered from than is the case with the cat.

Professor Hicks contended that in all this lay the value of the training, the frog as an animal for experiment giving the student an entirely erroneous idea of tissue viability and organic reaction. The course had been in operation for six years, and certainly at the time of its inception, if not still, was unique so far as the experience of a course in some thirty-five universities in Europe was concerned. Such a complete course was impossible in a large school, and this was the opportunity which attracted him to Adelaide.

After the intensive mammalian course the students carried out applied physiological investigation upon themselves. Their own venous blood was studied both for blood chemistry and for transport of oxygen and carbon dioxide. Artificial states of alkalosis and acidosis were induced and these studied on the human subject. Total metabolism in relation to fat, carbohydrate and protein was investigated by gas analysis of respired air, and basal metabolism was determined by the methods of Krogh, Haldane and Benedict. The students examined their own gastric contents and studied the effects of hindrance to respiration, such as would be produced in pneumonia, emphysema, asthma *et cetera*, and the respiratory mechanism, as well as the circulatory, was completely investigated. For example, students studied the cardiac output at rest and in exercise, and showed the difference in response of trained and untrained subjects. They carried out examinations such as those used for entrants to the air force, for life insurance *et cetera*.

In the laboratories students demonstrated methods of carrying out these examinations, and in the mammalian class room six operating groups were seen in action, demonstrating the following selected physiological features:

1. Effect of hæmorrhage recovery upon blood pressure and corpuscular volume. Comparison of saline and gum saline infusions in hæmorrhage.

2. Histamine shock and its effect on corpuscular volume in capillary blood. Effect of curative procedures.

3. Cardiac rhythm as influenced by depressor and accelerator reflexes, using the Broemsen manometer.

4. Pharmacology of adrenaline, pituitrin, amyl nitrite and acetyl choline.

5. Cardiometer showing volume changes with changes in rhythm.

6. Diuresis from isolated ureters, saline and sodium sulphate infusions and theobromine and urea being used.

The experiments were in no way especially staged for the occasion, but were done by the students without assistance and after they had been away from mammalian work for six weeks.

Research.

Research work was illustrated by a demonstration by Dr. R. F. Matters, showing the results of the past six years' work in connexion with female sex function. The preparation and standardization of œstrin and anterior pituitary hormone, which had been made and used clinically from this laboratory from the moment of their discoveries, was shown *in extenso*. Slides showed the effect of anterior pituitary hormone upon the cervical epithelium, and the results of diathermy upon human cervical overgrowth were exhibited. The collected results of some 300 Siddall and Aschheim-Zondek tests were demonstrated. Methods of investigating cerebro-spinal fluid for oxytocic principle were also demonstrated and tracings shown, proving the presence of such principle in the cerebro-spinal fluid of the parturient female. These were obtained with a rat's uterus standardized against pituitrin, and provided the only incontrovertible evidence of the phenomenon in man, as suggested by the work of Dixon and Marshall on the pig.

Mr. G. I. Cox gave a demonstration of his technique for the study of tissue-blood water balance, utilizing a highly accurate intravenous infusion pump designed and made in the department.

It was explained that the equipment with which the students worked, was made in the department, even including the double drum kymographs, the delicate adjustable Straub manometers and Deprez electro-magnetic signals. The present state of the equipment represented the cumulative results of five years' work, the first classes having carried on in the face of almost insurmountable difficulty.

NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Horsley, Charles Houston, M.B., B.S., 1930 (Univ. Sydney), 12, Edgeware Road, Enmore.

Lewis, David Herbert, M.B., B.S., 1927 (Univ. Sydney), Number 11, "St. Rowena," 37, Elizabeth Bay Road, Elizabeth Bay.

Correspondence.

LIPIODOL AND THE LUNGS.

SIR: Lipiodol can surely have gained no more enthusiastic advocate than Dr. J. F. Mackeddie. In support of its use in pulmonary diagnosis and therapeusis he has collected a formidable array of authorities and has presented a convincing case for lipiodol, which its critics would find difficulty in upsetting. With most of what Dr. Mackeddie says, I am in complete agreement. But an experience covering the past three years and resulting in the obtaining of well over two hundred bronchograms prompts me to discuss a few points in Dr. Mackeddie's paper.

In perhaps 90% of cases lipiodol is given to obtain an answer to one or both of the following questions: (i) Has the patient bronchiectasis? (ii) If so, is it unilateral or bilateral? In these cases the bronchiectasis, if present, is almost certainly basal. In technique I suggest, therefore, the following modifications, which I constantly employ now, except in the rare case where it is desired to fill the apex of the lung.

Chandler's trocar and cannula are employed; knife puncture is unnecessary. The patient is seated in the X ray room upright on a chair, with head against a pillow, which is propped by the wall or the side of the X ray table. When the lipiodol-filled syringe is attached to the cannula and an air bubble withdrawn to insure the cannula being in the trachea, the patient is tilted slightly to one side by an assistant holding the shoulders. Ten cubic centimetres are run into the bronchus on that side. The tilt is then reversed and the remaining half of the lipiodol flows into the opposite lung. The bases of both lungs are equally and entirely satisfactorily filled by this method, the right middle lobe usually filling as well. Should the patient cough slightly during the operation, the apices will also be illuminated, but this should not be sought, as once coughing commences, it may become uncontrollable and a skiagram cannot be taken. With a fibroid lung or possible carcinoma, for instance, the patient lies on his back and later is "turned to the desired side," but these are exceptions.

Further, no more than twenty cubic centimetres are required to give a perfect bilateral bronchogram, unless there chance to be cavities of considerable size, in which case up to forty cubic centimetres should be given.

In regard to tuberculosis, one cannot see how lipiodol would help in the diagnosis of this disease, nor does Figure VII offer convincing evidence. It can certainly exclude tuberculosis, however, by showing an extensive bronchiectasis to be the cause of the patient's signs and symptoms.

Dr. Mackeddie is hardly fair in his comment on the work of Armand-Delille and Moncrieff. Correctly, I think, they advise against the giving of lipiodol in frank tuberculosis. Their "signal success" was undoubtedly regarded by them as bronchiectasis in the first place, and their diagnosis established by a bronchogram. They did not give lipiodol to a patient with tuberculosis. In like fashion, on three occasions I have confuted the radiologist's diagnosis of pulmonary tuberculosis. In regard to the medium, I have used "Iodipin," "Oliolase," and "Hydriol" and have found them all to be irritating and cough-inducing, which lipiodol is not, unless it has been opened for a time, when it tends to assume the darker hue of its rivals and their tussigenic property.

If one were to criticize Dr. Mackeddie's illustrations, it would be on the score of their unilaterality. Thus Figure XVI shows an extensive saccular bronchiectasis of the right lung. The left, though practically unfilled, appears to show saccules at the base. Had it been better filled, gross disease might have been apparent. And yet we are told that "compression by air was found to be impossible." Should it have been attempted at all? Perhaps it were captious criticism to suggest that Figure IV does demonstrate bronchiectasis, whatever organisms were found in the sputum, or that the caption to Figure III is distinctly provocative, to say the least!

Dr. Mackeddie is to be congratulated for marshalling the uses of lipiodol in so impressive a fashion. It can undoubtedly be of the greatest value in the diagnosis of pulmonary disease, but it must be employed always in conjunction with a careful history and clinical examination of the patient, including sputum examination, and not as a short cut to diagnosis.

Yours, etc.,

COTTER HARVEY.

137, Macquarie Street,
Sydney,
November 9, 1931.

TRACHOMA AND DIET.

SIR: Owing to my absence from Australia, I have not, until now, had the opportunity of reading and replying to the letter of Dr. J. Lockhart Gibson, entitled "Trachoma and Diet," which you published in your issue of October 10, 1931.

Dr. Lockhart Gibson says in that letter: "I would suggest that trachoma is a food deficiency disease," and he infers that this suggestion has not been raised before. His references are to MacCallan and to Bishop Harman, both very eminent men, but both outside Australia. I am,

indeed, afraid that he must have confined his researches into records of overseas publications and have neglected the one so much nearer home, this journal.

In your issue of June 30, 1928, you published a paper of mine summarizing the treatment of trachoma, and in that paper I said: "There also appears to be a large vitamin element entering into the problem, for the more remote the district and the greater the difficulty in obtaining fresh greens, the greater does the incidence of trachoma seem to be" *et cetera.*

At the meeting of the Australasian Association for the Advancement of Science in Hobart in January, 1928, Dr. Harvey Sutton also referred to this aspect of my work, saying: "This finding seems to support the suggestion that trachoma is closely allied to conditions of poverty of diet, the result of remoteness from civilized centres."

Obviously, on this aspect of the matter, we arrived at these conclusions some years ago, and I am glad to be able to support Dr. Lockhart Gibson now.

I cannot agree with him, however, that trachoma is "not highly contagious": it may not be so in all its varying stages, but in the discharging phases, when it usually has a muco-purulent conjunctivitis superimposed, I feel sure it is.

In addition to vitamin lack, there are other predisposing factors tending to lower conjunctival resistance in trachomatous areas, factors like dust, heat and glare. These conditions, I feel sure, do not prevail at the Brisbane Hospital for Sick Children, and the fly of the controversy does not there get the same opportunities for carrying on its foul work.

My experience is confined to Australia, and particularly to its more remote areas, and I had no opportunity of seeing the troops overseas, to whom Dr. Lockhart Gibson refers. But is it not possible that the disease was prevented from attacking these men by the fact that they were under disciplinary medical care during the whole of their stay there (like the children in the Brisbane Hospital for Sick Children), and not because of the forbearance of the Egyptian and Mediterranean flies. The ubiquitous fly of the trachomatous areas cannot so easily be relieved of its burden of opprobrium.

With regard to the diminished prevalence of the disease, Dr. Lockhart Gibson says he has heard of no explanation more plausible than that of improvement of diet. While agreeing on the diet question, might I suggest to him that the more plausible explanation is to be found in a survey of the work of the education departments of Australia, with their constant insistence on treatment, prophylaxis and fly destruction.

I shall be very pleased to forward to Dr. Lockhart Gibson a reprint of the paper I have referred to, if he would care to have it.

Yours, etc.,

CAWLEY MADDEN, M.B., Ch.M.

Sydney,
November 6, 1931.

Bibliography.

Cawley Madden: "Notes on the Diagnosis and Treatment of Trachoma," THE MEDICAL JOURNAL OF AUSTRALIA, June 30, 1928, page 800.

Harvey Sutton: "Transactions of Australasian Association for Advancement of Science," Hobart, 1928, Volume XIX, page 482.

ORTHOPÆDIC SURGEONS.

SIR: I have attentively read Dr. Hoets's letter in your issue of October 31. My subject all along has been "bad back." Dr. Hoets's subject has been myself (no importance), except for the following categorical general affirmative in his last paragraph: "Orthopædic surgeons are fully alive to the 'psychological handicaps' from which many patients suffer."

Shall we call it rhetoric?

Yours, etc.,

C. E. CORLETTE.

Sydney,
November 7, 1931.

CHRISTIAN SCIENCE.

SIR: The statement is being made that cases of cancer, tuberculosis, Bright's disease, diabetes and osteoarthritis are being cured in Sydney by Christian Science. Now, if this is so, there are surely some medical men who at one time or other have had these cases under observation and should be able to testify as to what has happened.

Will they not come forward and tell us what the facts are?

Yours, etc.,

"M.D."

Sydney,
November 4, 1931.

TRAUMA AND ORGANIC VISCERAL DISEASE.

SIR: I have read with much interest the paper contributed by Dr. C. E. Corlette on trauma in relation to organic visceral disease. I was sorry that I was unable to attend the meeting at which it was read, as I was desirous of taking part in the discussion and of making special reference to one of the compensation cases to which he makes special reference: *Ryman versus Municipal Council of Mosman.*

As one of the three general practitioners involved in this case, I wish to protest against any reference to the case unless the whole question of the causation of internal hernia had been referred to, and also to protest against the gibe at the opinions of other medical men without producing any evidence to show their falsity.

I am still more concerned to open up this case, since not only is there a question of individual injustice involved, but the establishment of a false precedent.

The salient features of the case are as follows: An employee of the Mosman Municipal Council was engaged in lifting large sandstone blocks from a platform on which he was standing, to the top of a wall, the height being five to six feet. He was seized with sudden abdominal pain, which led to vomiting. At operation twenty-four hours later coils of small bowel were found incarcerated under old adhesions and matted together with recent plastic lymph. The three medical practitioners concerned gave evidence that in their opinion the immediate cause of the incarceration was the strenuous muscular effort and the pressure of the contracted abdominal muscles.

The insurance company had an array of five medical witnesses bearing the *cachet* of Macquarie Street. I heard the evidence of the first of these. He stated that the condition found at operation, as described, could not possibly have been caused by any muscular effort. He reiterated "it was impossible." This opinion was concurred in by all five witnesses, I have been informed.

Naturally, on the weight of evidence an adverse verdict was given.

I wish to invite a discussion on the causation of internal hernia, and invite the opinions of other "specialists" on the relationship of muscular effort to these cases.

Yours faithfully,

F. S. STUCKEY.

Mosman,
New South Wales,
November 8, 1931.

CÆSAREAN SECTION.

SIR: From time to time a wave of enthusiasm for the performance of Cæsarean section sweeps over the profession. Plausible arguments are put forward in its justification. An allegedly low mortality, 1% to 2%, is frequently claimed, although a wide employment of the operation takes a greater toll than that. The surgical and obstetrical sequelæ cannot be ignored. A Cæsarean section, in the absence of strong indications, too often leaves a mutilated woman. In America the widest indications have been used, and Whitridge Williams remarks that:

> It has a 10% mortality in such circumstances, which means that many thousands of women are dying of

Cæsarean section who ought not to die. At the present time I consider that the operation is being abused and that not a few patients are sacrificed to the "*furor operativus*" of obstetricians and general surgeons who are ignorant of the fundamental principles of the obstetric art. In my experience as a teacher, the hardest thing I have to do is to hold the men down and keep them from unnecessary procedures.

Among others, Gilbert Fitzpatrick says:

> The fact of the great extension of section deliveries is an indictment of the inadequacy of obstetric knowledge and an indication of obstetric failure.

A. K. Paine:

> Cæsarean section is recognized as having in itself the highest maternal mortality of any delivery operation. The obstetrician faces a duty and a moral obligation to rise superior to expediency in his conduct of a delivery.

Gauss:

> Operative procedures introduce new dangers for the mother. The principal improvements in technique have as their basis an earlier time for operation, which means at a time when a sufficiently founded indication often really cannot be made. To evaluate fully all the sequelæ of Cæsarean section one must consider also the morbidity following the operation, bronchopneumonia, thrombosis, embolism, wound infection, peritonitis, ileus, endometrioma *et cetera*. A further drawback is the diminished safety for subsequent deliveries, or the voluntary or necessary sterility subsequent to the section.

Hellmuth:

> I conclude from the mortality and numerous sequelæ following Cæsarean section that the indications for the operation should be very strictly drawn. The most frequent and unquestioned indication is afforded by serious disproportion between the fœtal head and maternal pelvis.

Your correspondent considers that doubtful cases of disproportion in *primiparæ* should always have the benefit of a trial labour. How often do we find a floating head in a *primipara* before and early in labour successfully descend in spite of our apprehensions? In *placenta prævia centralis* Cæsarean section has a place, particularly in the elderly *primipara*. Whitridge Williams says, however, that if Cæsarean section were generally employed in *placenta prævia*, the maternal mortality would be increased. My own view is that Cæsarean section is a great servant, but an exacting master. While, no doubt, its judicious use often saves life, a widening of indications is always threatening us, and is a menace to womanhood, particularly in this country. I feel that better and still better obstetric teaching is the road to progress in obstetrics. The loosening of control over the performance of Cæsarean section can only lead to imprudent operating and increased maternal mortality.

Yours, etc.,

ROLAND BEARD,
Gynæcologist, Adelaide Hospital.

North Terrace,
Adelaide,
November 10, 1931.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on November 2, 1931.

The degree of Doctor of Medicine (M.D.) was conferred in person upon Michael Richard Flynn, B.A., B.Sc., M.B., Ch.M., and John Kempson Maddox, M.B., Ch.M.

The appointment of the following gentlemen as Honorary Assistant Clinical Tutors in Medicine in the teaching hospitals named was approved:

Royal Prince Alfred Hospital: Dr. R. S. Steel, Dr. D. M. Ross, Dr. I. A. Brodziak, Dr. P. J. Markel.

Sydney Hospital: Dr. W. E. Fisher, Dr. H. M. Owen.

Saint Vincent's Hospital: Dr. J. D. Maude.

Medical Practice.

INCOME TAX.

THE Council of the Victorian Branch of the British Medical Association has forwarded for publication the following opinions given to Sir James Barrett in regard to income tax.

Income Tax Office,
October 27, 1926.

Sir James Barrett,
105 Collins Street.

Dear Sir,

INCOME TAX ACTS.

Referring to your letters of the 7th and 25th inst., I desire to advise that where medical books are destroyed or become too dilapidated for further use the cost of replacement up to the original cost of the book replaced may be claimed as a deduction in the State assessment.

The cost of any additional books to those already on hand would not be a permissible deduction.

If the medical periodicals are of lasting value and not merely containing current information, the value may be included in those of other medical books of your library.

On receipt of this information an amendment will issue.

Yours faithfully,
(*Signed*) R. M. WELDON.

September 30, 1926.

If the value of medical books be furnished, the matter of allowance for depreciation will receive attention. The maximum amount allowable being 1 per cent. The allowance of £10 for medical and other periodicals is only an estimate of the cost and is not incorporated in the Act, but where a taxpayer submits evidence that a greater sum has been expended, the full amount will be allowed.

September 7, 1926.

For State purposes replacement may be claimed when effected.

Proceedings of the Australian Medical Boards.

QUEENSLAND.

THE undermentioned have been registered under the provisions of *The Medical Act of* 1925 as duly qualified medical practitioners:

Roberts, William Henry, M.B., Ch.M., 1916 (Univ. Sydney), Brisbane.

Holt, John Ackland, M.B., Ch.M., 1925 (Univ. Sydney), Wynnum South.

Borch, Rudolph Hermann von der, M.B., B.S., 1926 (Univ. Adelaide), Mount Isa.

Restoration to the Register:

Hackett, Henry William, L.R.C.P. and S. (Ireland), L.M., 1917, Townsville.

Obituary.

WALTER CRESSWELL HOWLE.

We regret to announce the death of Dr. Walter Cresswell Howle, which occurred on November 15, 1931, at Sydney, New South Wales.

Books Received.

MANIPULATION AS A CURATIVE FACTOR; OSTEOPATHY AND MEDICINE, WITH AN APPENDIX ON HAY FEVER, by E. Mellor; 1931. London: Methuen and Company, Limited. Demy 8vo., pp. 268, with illustrations. Price: 10s. 6d. net.

THE THYROID AND MANGANESE TREATMENT: ITS HISTORY, PROGRESS AND POSSIBILITIES; THE INTRODUCTION OF A METHOD OF SYSTEMIC DETOXICATION IN THE CONTROL AND PREVENTION OF DISEASE, by H. W. Nott, M.R.C.S., L.R.C.P.; 1931. London: William Heinemann (Medical Books) Limited. Crown 8vo., pp. 280. Price: 7s. 6d. net.

Diary for the Month.

Nov. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 25.—Victorian Branch, B.M.A.: Council.
Nov. 26.—South Australian Branch, B.M.A.: Branch.
Nov. 26.—New South Wales Branch, B.M.A.: Branch.
Nov. 27.—Queensland Branch, B.M.A.: Council.
Dec. 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Dec. 1.—New South Wales Branch, B.M.A.: Ethics Committee.
Dec. 2.—Victorian Branch, B.M.A.: Council.
Dec. 2.—Victorian Branch, B.M.A.: Annual General Meeting.
Dec. 3.—South Australian Branch, B.M.A.: Council.
Dec. 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Dec. 10.—New South Wales Branch, B.M.A.: Branch.
Dec. 11.—Queensland Branch, B.M.A.: Branch (Annual).
Dec. 15.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Dec 18.—Queensland Branch, B.M.A.: Council.

Medical Appointments.

Dr. G. S. S. Hayes (B.M.A.) has been appointed Medical Officer for Venereal Diseases, Department of Public Health, Brisbane, Queensland, pursuant to the provisions of *The Health Acts*, 1900 *to* 1922.

* * *

Dr. W. N. Robertson (B.M.A.) has been appointed to the Brisbane and South Coast Hospitals Board, Queensland, as a Government Representative, pursuant to the provisions of *The Hospitals Acts*, 1923 *to* 1929.

* * *

Dr. I. G. M. Halley (B.M.A.) and Dr. J. S. Proctor (B.M.A.) have been appointed as Official Visitors to the Mental Hospital at Parkside, South Australia, under the provisions of the *Mental Defectives Act*, 1913.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xviii.

BALMAIN AND DISTRICT HOSPITAL, SYDNEY, NEW SOUTH WALES: Assistant Surgeon.
FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Junior Resident Medical Officer.
INSPECTOR-GENERAL OF HOSPITALS DEPARTMENT, ADELAIDE, SOUTH AUSTRALIA: Honorary Surgeon.
THE OTAGO HOSPITAL BOARD, DUNEDIN, NEW ZEALAND: Resident Surgical Officer (Senior), Resident Medical Officer (Senior).

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL, are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Mines. Toowoomba Associated Friendly Societies' Medical Institute.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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